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Original Articles and Clinical Cases.

HYSTERIA AND NEURASTHENIA.

BY SEYMOUR J. SHARKEY, M.A., M.D.OXON., F.R.C.P.

Presidential Address, delivered before the Neurological Society of the United Kingdom, on February 4, 1904.

GENTLEMEN,—On taking the chair of the Neurological Society I return you my most hearty thanks for electing me to so distinguished a position, though I cannot but feel that my pleasure at the honour conferred is tempered by a consideration of the difficulty of fulfilling the duties among so many eminent scientific men. I rely, however, upon the continuation of that indulgence of which you have already given evidence in electing me your President. It is customary for the President on taking the chair to address the Society on some subject of interest, and the selection of such a subject has been to me no easy matter; for the demands made upon the energies and time of a physician attached to a large hospital make it impossible for him to carry on physiological and pathological research, or even to keep abreast of the knowledge and fluctuating opinions of the day. It is difficult, under such circumstances, to submit to a society containing so many members actively engaged in scientific research, anything which they will deem worthy of the occasion. The practising physician can hardly have

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recourse to that upon which they rely for clearing up doubtful points and extending the boundaries of knowledge, viz., experiment. What value, therefore, will they attach to anything which may be said when it lacks the solid foundation upon which they are wont to build? I believe that it would be of the greatest advantage for the advancement of knowledge, and would yield the most valuable suggestions as regards the direction of experimental and other physiological work, if physiologists could go round the wards of a hospital regularly, even for a few hours a week. Amongst other things, they would see that there are certain subjects which can hardly be dealt with experimentally. Experiments on the cord and brain are on somewhat the same level as those performed by gross disease, with this exception, that the physiologist can perform his experiments when he will, the physician has to wait patiently for years to observe the effects of some experiment he wishes to see performed by disease, and even then it is hardly ever precisely what he desired; but in both cases alike the process is a rough one. Finer experiments, however, are carried out in those cases of disease where the functions of organs are altered without any gross, tangible change of structure presenting itself. These the experimentalist cannot conjure up at will, and they can only be observed by those who have to deal with the ailments of their fellow-men and women. Such diseases are generally termed "functional" diseases. Whilst gross diseases of the nervous system force themselves most upon the attention, and are the cause of a great number of deaths, I do not think I am wrong when I say that the effects of the ill-health, suffering, and incapacity produced by functional diseases are even more serious and far-reaching. I propose, therefore, to offer the Society a few remarks upon this subject. The scope of them, however, must needs be very limited, for the diseases themselves affect every part of the nervous system and involve the whole subject of nerve physiology and pathology, while in the short space of time allotted to me but few of them can be touched upon.

One of the commonest of these functional affections is

HYSTERIA. In considering the physiology of man, or of any other class of animals, physiologists are liable to suppose that individuals may be looked upon as practically alike, though their attention must often be drawn to the fact that they react somewhat differently under observation. To the physician one of the most striking things is the difference between individuals, and this difference is one of the important factors in disease. We see this strikingly illustrated in families where, though the several members are much like those of other healthy families, in the course of their lives they show that they are not the same. They are born different; so that we may see, for instance, the arteries in one family slowly but surely degenerate, while those of another family retain their healthy peculiarities to an advanced age. In other families the kidneys degenerate early, or the gastro-intestinal canal shows itself incapable of carrying on the digestive processes in the steady, unerring way that one observes in others. These differences are, no doubt, due to the original laying down of the several systems and to the inherited conditions of their cellular constituents. There is no part which illustrates these observations better than the nervous system. One portion of a liver or a kidney is much like another, and the whole organ consists of an agglomeration of similar lobes and lobules, which are very like those in lower animals. But the nervous system of man, and to a less extent that of animals, consists of a great variety of sub-divisions and systems which are not alike. And in the ascending scale of living beings new and higher parts are added. This increasing complexity of the nervous system reaches its maximum in man. The stability of any portion of the nervous system, that is to say, its power of resisting causes which derange function, is, other things equal, proportional to the length of time during which it appears to have existed as a part of the nervous system. In man the cerebral cortex is the part which has been most recently developed, and one might expect that it would frequently show signs of functional disturbance. But even amongst the apparently healthy there are the greatest differences as

regards stability, so that the limits of health and disease are not capable of being rigidly defined. It requires a certain degree of adverse conditions to overthrow the equilibrium even of the unstable, and the latter may escape such unfavourable circumstances and so never betray their weakness.

The most active constituents of the cortex, physiologically, are the nerve-cells, and it is probable that they are the main seats of functional derangement; but we cannot be sure that the rate and ease of conduction in their afferent and efferent fibres may not be important factors in nerve derangement.

In hysteria the brain is probably the part of the nervous system which is most affected. This view is supported by such considerations as the following:—

When anæsthesia occurs in islets of the skin its distribution does not correspond with the distribution either of peripheral nerves or of blood-vessels.

Pitres reports a case in which there was hemianæsthesia, and when the ulnar nerve was pierced with a pin no pain was felt, although contraction of muscles supplied by it occurred, showing that the nerve retained its conductivity, and that the affection was therefore not peripheral. Indeed, hemianæsthesia, which is of frequent occurrence in hysteria, would be anatomically inexplicable on any other supposition than that it was of cerebral origin.

It is a curious fact that when hemianæsthesia due to gross disease occurs the patient is conscious of his loss of sensation, but in hysteria, as a rule, the patient is not aware of it. In the former cases the sensory fibres in the internal capsule are generally diseased, while the cortical centres are not. The centres are therefore conscious of the loss, and I would suggest as a reason why the hysterical patient is not conscious of it is because the cortical centres are the parts affected.

It is well known that hysterical patients very easily acquire conditions, such as rhythmical and other spasms, from association with patients who are the subjects of them. This must be caused by the idea, uncontrolled, giving rise

to the same spasm; it is, in fact, a case of ideational automatism, and of cortical origin. If the view that the cerebral cortex is the seat of functional derangement in hysteria is correct it accounts for the great variety of symptoms met with in the disease. For not only are general and special sensation, voluntary movements, speech, emotion, &c., arranged for in the cortex, but the action of the heart, respiration and other processes which may be deranged in hysteria, are at least influenced by cortical action.

Wherein, then, lies the difference between the normally acting brain and the hysterical? Many hysterical people are bright, intellectual and attractive. But their brain action is characterised by three peculiarities at least: (1) Explosiveness, if I may say so; (2) want of control; (3) proneness to exhaustion. So that two sets of phenomena occur: one a too active and uncontrolled nerve discharge, the other paralysis of various kinds, sensory and motor.

Emotion, for example, in a healthy person initiates muscular movements of various kinds, expressions of joy or sorrow, as the case may be, controlled, however, within appropriate limits; while the same cause of emotion may, in the hysterical subject, reverberate impetuously and unrestrainedly through the motor centres, mingling the natural expressions of joy and sorrow confusedly together, and producing irregular movements of the limbs, or even general convulsions.

In other cases emotion inhibits nerve action and produces various forms of sensory and motor paralysis. Or the latter may be due, not to inhibition, but to exhaustion of cells of the cerebral cortex.

The exciting or restraining influence may be central, such as an emotion or an idea, but it may also be peripheral, as is shown by the various points, whether superficially or deeply situated, pressure upon which either gives rise to a hysterical fit or stops one which has already commenced.

The explanation of muscular spasm and of sensory and motor paralyses in hysteria is rendered very difficult by the fact that parts of the brain can certainly inhibit nerve

centres lower down. This is shown by the increased action of the cord on section of the pyramidal tracts. In the case of a limb being paralysed, therefore, two reasons may be suggested for this: one that the cells in the cord are inhibited, the other that part of the motor centres of the cortex are paralysed. The latter is the most probable hypothesis, for the deep reflexes of the paralysed limbs are usually increased, showing that the cells of the cord are over-active and not inhibited. Paralysis with increased deep reflexes is often the first stage of paralysis with spasm, so that where the latter is present in hysteria it may be explained by a more complete paralysis of the cortical centres.

There is a curious contrast between the hemiplegia of hysteria and that due to gross disease. In the latter the arm is usually most paralysed, the leg less than the arm, and the face least of all. In hysteria the leg is generally the most affected, the arm less, and the face not at all. I would ask the physiologists how this is to be explained. The explanation which occurs to me is this: If the hemiplegia is due to the cortical motor cells being inactive or paretic, then one would expect that the parts most distant from these centres would be most powerless, and those least removed from them less so, or unaffected. For nerve force generated in the cells of the cortex would only be propagated to the distant muscles of the leg if considerable, though it might, if far less, reach the nearer muscles of the face and tongue. In gross disease the reason why the leg is least affected is that parts which are usually associated in action on the two sides of the body are intimately connected in their nerve supply, so that the centres can supplement each other should one side be paralysed. But in the case of the hysterical patient there is evidence to show that though one hemisphere may be so affected as to produce hemiplegia the other is also affected, though in a less degree, and so cannot make good the defect of its fellow.

Another curious point about hysterical affections is this, the frequency with which anæsthesia occurs, unilateral, bilateral, or disseminated in islets. In gross disease of the brain hemianæsthesia is rare, in hysteria quite common.

When it does occur in the former it is almost always accompanied by hemiplegia: in the latter it may be present in a marked form without hemiplegia. In gross disease its cause is direct injury to the posterior sensory fibres of the internal capsule, and as they are in close connection with the motor strands, the two are almost invariably paralysed together. But above the capsule the fibres separate to end in different regions of the cortex; and here they are probably widely separated from each other, so that a functional change in one part of the cortex, in hysteria, might easily occur without other parts being involved. In gross disease of the more distant parts of the nervous system, the cord for instance, anæsthesia is comparatively difficult to produce, and it recovers before the motor paralysis. So that one never finds the lower limbs anæsthetic from cord disease after the motor paralysis has passed off, whereas anæsthesia of a marked kind often occurs in hysteria without any motor palsy. There is probably more than one path by which sensation from the periphery of the body can make its way to the cortex which receives and appreciates it, so that it requires very complete destruction of the cord to stop the passage of these nerve impulses from the limbs. But in hysteria it is the perceiving cortex itself which is insensitive, and no recovery can occur until it resumes its function. The dissemination of islets of anæsthesia over the body in hysteria without any necessary motor paralysis has its explanation in the fact that the sensory fibres leaving the internal capsule break up and end separately in the cortex, and therefore small cortical areas, if functionally depressed, would give rise to limited areas of sensory deficiency in the skin. But in gross disease the sensory fibres are pretty closely packed in the brain and cord, so that even a limited focus of disease would affect a large number, and where they spread out again at the periphery of the body, just as they do in the cortex above, they are associated with motor nerves, and could therefore rarely be paralysed alone by disease.

The anæsthesia in hysteria may be complete or only affect certain forms of sensation. Analgesia may occur, or

thermoanæsthesia or electroanæsthesia, or alterations in muscle sense, but tactile sensation is the last to go, and never disappears alone. The explanation of this is uncertain. In disease of the distant peripherally distributed sensory nerve-fibres it is much more easily affected, for if the receiving terminals are paralysed no external impressions can enter upon the upward path to the cerebral centres, however healthy the latter.

In the cortex itself, if one may be allowed to speak of the "field of sensation" as one does of the field of vision, the area of tactile sensation may perhaps be compared with the yellow spot in the retina, and both may be looked upon as anatomically constructed to be the parts most sensitive to impressions, each in its own field. And just as there may be a dissociation of sensations, certain forms—such as those of pain and temperature—disappearing while tactile sensation remains, so in the eye vision may be retained, while colour vision may be altered, violet and green disappearing first, and blue, yellow and red later. Such points as these, however, must be left to the physiologists to explain.

But the visual changes mentioned are not the only ones which occur in hysteria, nor, indeed, are they the most important. The most characteristic alteration is the concentric contraction of the fields of vision. So marked may this be that the fields may be reduced to very small circles around the yellow spots. In one sense the change is symmetrical, that is to say, it occurs in both eyes; but in another sense it is generally not so. If a hysterical patient has, for instance, hemianæsthesia of the left side of the body, this includes vision and hearing too. But the change in the fields of vision is not like that which occurs in hemianæsthesia from gross lesions; it does not consist of hemianopsia, but of a concentric retraction of the field around the yellow spot. It is doubtful whether hemianopsia ever occurs in hysteria; I have never seen it myself. But whether it occurs or not, concentric retraction of the fields is the change which occurs almost invariably. On the hemianæsthetic side, however, the contraction is far greater than on the other.

Now, I would ask the physiologists how this state of things can be explained. It hardly tallies with the view that the homonymous lateral halves of the retinæ are in relation with a half vision centre in the corresponding occipital lobe, and that each hemisphere has no more intimate relation with the retina on the opposite side than with that on its own side. The dynamic changes which occur in the brain in hysteria are no doubt bilateral, and do not, as a rule, confine themselves to one hemisphere only, though they affect one more than the other. But if it be assumed that the retina, on each side, while it is in connection through the occipital half vision centres with both hemispheres has also a more direct connection with the hemisphere of the opposite side, this would explain how the field of vision is most contracted on the side of the body on which the other sensations are paralysed. A different explanation of the phenomenon is, however, possible. The contraction of the fields of vision may be a peripheral change and not a cortical one. The retina is well known to be less and less sensitive as one passes from the yellow spot to the periphery ; and consequently if there are changes which act in a paralysing manner upon the distribution of the optic nerves, the effect will naturally show itself most where the function of the retina is normally least vigorous ; while the function of the most sensitive parts about the yellow spot will be the last to be extinguished. But there is no good reason for looking upon hysteria as a disease of peripheral nerves; it is a disease principally of nerve centres. We must, however, remember that the retina in its origin is really a process from the brain, and not strictly speaking a peripheral nerve.

The occurrence of anæsthesia in hysteria is not confined to the skin and special senses, the mucous membranes and deeper tissues may also be affected. Anæsthesia of the pharynx, for instance, is of great importance in the diagnosis of the disease ; and Pitres asserts that patients often tolerate, and even enjoy, the hardest pressure that can be applied to the epigastric region. Even the periosteum and bones may be analgesic. Sometimes, on the contrary,

hyperalgesia is complained of, and pain of the most constant and stubborn kind is experienced. Some of the most characteristic positions for it are the vertex, the spine, the coccyx, and the left iliac fossa. But it occurs in many other positions, too.

While it is upon the brain that the brunt of the disease falls in cases of hysteria, it is probable that the nerve centres in the spinal cord may also be affected, either directly or indirectly. As far as these centres are concerned inhibition is probably mainly exerted by the pyramidal tract. In health a steady stream of nerve force descends along it from the cortical centres and controls the activity of the spinal centres, and when an interruption of these strands occurs through gross disease a hyperactive condition of the motor cells of the cord results, shown by increased reflexes, and sometimes by contraction of the limbs. If, as we suppose, the nerve force generated in the cerebral cortex in hysteria, though discharging occasionally with spasmodic violence, is not sufficient to cause a steady, controlling influence to pass down the pyramidal tracts the centres in the cord would be in a condition of hyperactivity, shown by marked increase in the reflexes. This generally is so. Very slight further diminution in the inhibition exerted by the pyramidal tract, together with some peripheral irritation, might easily produce reflex spasm of the limbs; in fact, the condition would be what Pitres describes as "l'état d'imminence de contracture," which, he says, exists in the majority of confirmed cases. A good instance of this is the following: A girl, aged 25, previously healthy, was found to have a cystic tumour of the left ovary. Dr. John Phillips removed it, and at the operation found that it was perfectly free and not adherent to any of the structures around. Some little time afterwards I was asked by him to see her for spasm of the adductors of the thigh on the left side and inability to support herself on the left leg in walking. I found that the slightest touch of the skin over the adductors produced immediately great increase of the spasm. The case was shown to be one of hysterical spasm by the following facts. She had very much increased knee jerks, most

marked on the left side, absence of plantar and pharyngeal reflex, marked analgesia of the left leg and the left half of the body, diminishing from below upwards and sparing the face.

But it is probable that the functional inertia of nerve-cells is sometimes present in the cord of such cases as well as in the brain, for some of them have flaccid paralysis of limbs and absent plantar reflex, although another explanation is possible, viz., that the pyramidal tract is over-acting and so inhibiting the reflexes of the cord. It may be objected that one cannot explain both hyperactivity of the cord cells and inertia as well by supposing that in the same disease the pyramidal strands are sometimes over and sometimes under-acting. But this very uncertainty in the action of nerve centres, which is sometimes exaggerated and sometimes depressed, even in the same case, is one of the great characteristics of hysteria. Hence arise the various forms of hyperæsthesia and spasm on the one hand, and anæsthesia and paralysis on the other. In a healthy person, with normal, stable nervous system, there is an equable, steady current of nerve force, passing from centre to centre, resulting in the production of a state of equilibrium and healthy, mutual control. In hysteria there seems to be diminished pressure, if I may say so, so that energy ebbs and flows, and centres sometimes exhibit explosions of activity, and sometimes become almost inert. The activity of the latter may, however, suddenly revive and a paralysis of movement or sensation disappears, owing to various causes, such as an access of anger, joy, fright, &c. The sudden transition from inactivity to activity shows that there can be no fundamental change in the cells, but only a dynamic one.

How do such redistributions of nerve energy arise? Can it be that there are certain cells amongst the millions which exist in the layers of the cortex the function of which is to generate nerve force for distribution to the various centres? It is certain that the exhaustion of some regions entails the exhaustion of others which have not been in activity at the same time; intellectual work, for instance, causes brain

fatigue which renders physical work difficult, and *vice versa*. This suggests that there is either a common source of energy on which the various regions of the cortex draw when in active work, or else that nerve force can flow from one to another, or perhaps, as others suggest, that the fatigue products of centres in action act detrimentally on the rest. Whatever be the explanation, in hysteria some centres may retain their usual activity, while others are inactive, and produce various forms of paralysis, while the extent and intensity of the loss of function may vary almost from day to day.

In the Gulstonian Lectures, which I delivered in 1886, I wrote as follows: "Some think the centres are starved by contraction of their nutrient blood-vessels. It seems to me more likely that the nerve centres are exhausted by their own action. Thus, sudden emotional excitement may bring on hysterical phenomena indicating nerve exhaustion, such as hemianæsthesia, paralysis, or spasm. It is important to remember that the exhaustion of one area seems to affect the whole system. This may indicate that the various differentiated centres draw force from some common supply. Considering the enormous number of cells which are found in the cerebral gray matter, many without evident connections, is it going too far to suppose that a large number of these are factories for the production of nervous energy which is necessary for the continuous and regular action of the highest centres, and which flows in increasing quantities towards those centres which are from time to time brought into action? Constant supply would mean constant power in all centres, and, as the latter are bound together so as to produce a physiological equilibrium, constant power would involve constant control of one by the other.

"When the store of constantly produced force (represented, of course, by structural peculiarities) is by inheritance small, or when it has been exhausted by unnatural calls upon it, there would be insufficient to supply all the centres, and those to which the nerve paths are most freely open by habit would command it, while others would be starved, and loss of equilibrium, or the natural control of

one centre by the healthy action of others, would be the inevitable result. If we do not suppose any such reservoirs of nerve energy, how can we explain the exhaustion of all by the excessive action of some centres? Why is a person who has had excessive mental work less able for the time to do active bodily work? And why when bodily fatigued are we less able to do intellectual work?"

In BRAIN, Part II., 1903, Dr. McDougall has a paper on "The Nature of Inhibitory Processes within the Nervous System," in which he develops a similar idea. Here are some quotations from his article: "Inhibitory effects in the nervous system result from a process of competition between the inhibitory and the inhibited tract, the former draining to itself a part of the energy liberated in the latter." "Inhibition appears always as the negative or complementary result of a process of increased excitation in some other part." "The mind has only a limited quantity of energy, which will not suffice for the simultaneous maintenance of two mental processes." "Inhibition is due to competition among nerve paths for the living energy present at the moment in the nervous system." "The relation of reciprocal inhibition obtaining between all the different organised neural systems, constituting paths of forward conduction in the higher brain levels, is due, I suggest, to the fact that they all drain one common store of neurin contained and constantly generated in the interconnected mass of afferent neurones, and seeking constantly to escape by the paths of least resistance into motor neurones, and so into the muscles."

Amongst the hysterical manifestations which occur in such variety are some which suggest abnormal conditions of centres in the medulla oblongata or in the sympathetic system.

Well-marked cases occur in which respiration and circulation are affected. The movements of respiration may be slowed or accelerated, and so may the heart's pulsations. The following is a remarkable instance:—

A girl, age 21, began in May, 1886, without evident cause, to have a feeling of fainting, or "catching in her breathing," when

she laughed or cried. She got worse and worse, her breath shorter and respiration more rapid, until she was confined to bed ten months before I saw her, and had remained there ever since. For two months she was sick after everything she took, and, though this trouble then passed off, she had taken very little nourishment during her whole illness. What food she had taken gave her no discomfort. Her bowels and catamenia had been regular. She had lost much flesh. When I saw her I found her propped straight up with pillows, and breathing very rapidly. Respirations then were about 120 a minute. On my asking whether it was often so, both the mother and doctor said this had been going on for ten months. The mother further asserted that it remained the same whether strangers were there or not, and that she never noticed much difference. The lips were not blue, but the nares dilated regularly with each inspiration, and the latter was accompanied by the sound usually present in great dyspnœa. The pulse was 130 and there were no murmurs over the heart or other signs of disease.

This patient was isolated and treated with massage, full feeding, &c., and got quite well. Subsequently she trained at a large London hospital, and has been an active nurse ever since.

It might be supposed that such phenomena as these resulted from derangements of the functions of the medulla. But they can be produced by stimulation of the cerebral cortex. It is doubtful whether the conditions even of vaso-constriction and vaso-dilatation are due directly to any sympathetic changes, for such phenomena can be produced by stimulation of the cortex of the brain. It is a frequent observation that on pricking the anæsthetic cutaneous surface of a hysterical patient no bleeding occurs. Similarly local œdema is present in some cases, due probably to dilatation of the vessels.

Moreover, after the disturbance of the brain which produces a hysterical fit has passed off, there is a great increase in the amount of urine passed, which probably depends on dilatation of the renal vessels, causing flushing of the kidney. Sometimes, on the contrary, hysterical anuria may occur and last for days, indicating a contraction of the blood-vessels of the kidney. Spontaneous hæmorrhages in the skin are

likewise occasionally observed, due, it may be presumed, to extreme dilatation of vessels.

Hysteria, then, we must look upon as a condition in which the brain, although anatomically not to be distinguished from that of healthy people, is physiologically different. It is more excitable; the nerve discharges are more violent, and more easily produced; less under control. The nerve force ebbs and flows among the centres in such a way that certain parts are left high and dry, so to say, while others are flooded. And these conditions are liable to rapid fluctuations, so that symptoms appear and disappear in a remarkable manner. This condition of brain is probably congenital, and, as Pitres says, "En réalité on nait hystérique, on ne le devient pas." But patients who are born so need not necessarily suffer from palpable symptoms, though they are always in danger of doing so. They may, nevertheless, betray themselves to the careful observer by the presence of so-called "stigmata," *e.g.*, anæsthesia, of which the patient is unaware, absence of the pharyngeal reflex, contraction of the fields of vision, markedly increased deep reflexes, abolition of plantar, or other superficial reflexes, &c. As Professor Obersteiner says, neither molecular changes in the nervous system nor disordered nutrition are satisfactory explanations of symptoms which may come and go with such rapidity. The patient may present a condition of blooming health, while the most far-reaching alterations of nutrition may occur in other diseases without producing any symptoms of hysteria.

Nor do vascular changes offer a solution of the problem, for the anæsthetic conditions which occur in the skin do not follow the distribution of vessels, and it is still doubtful whether there are any vasomotor nerves in the brain. The condition appears to be a dynamic one, which we do not at present understand. Nor does the theory of neurones help us much, even if it were proved that the cell processes are capable of retraction from, or approximation to, those of their neighbours. We must look to the physiologists for an explanation of the normal cerebral functions before we can hope to interpret the abnormal changes which occur

in hysteria and allied conditions. But whether we clearly understand the abnormal processes going on in the nervous system in hysteria or not we have to treat the victims of it to the best of our ability.

If the disease be looked upon as a congenital condition we cannot hope to cure it, that would involve rebuilding the nervous system *ab initio*. But, as has been already remarked, latent stigmata may be the only evidences of the condition, and further manifestations which seriously interfere with the uses and pleasures of life may be absent. Methods of prevention and those directed to cure must follow similar lines. Broadly speaking, the well-known laws of health must be observed, and a quiet, pleasurable, contented life must be secured if possible. When serious symptoms of the disease have already manifested themselves the recovery of losses in general nutrition may be greatly assisted by massage, if accompanied by rest. But massage may positively aggravate the disease if it is employed while the patient pursues the same exhausting life which has led to the breakdown. It is then only adding one more cause of exhaustion. In most instances rest in bed for some weeks — six to eight — is required. The upsetting effects of emotions, whether of joy, or still more of sorrow, and the worries of every-day life, have a much greater effect upon hysterical people than upon the healthy, therefore to save them from these isolation is requisite; but unless great care is taken this may do the patients more harm than good. It is not uncommon to find that medical men, having correctly diagnosed a case as hysteria, and having also rightly recommended isolation, massage, electricity and good feeding, send for a nurse to take charge of the patient, without taking any serious thought whether the two will make suitable companions. And yet this is the most important point in the whole treatment. One sees the effects upon ordinary healthy people of constant association with an uncongenial companion; but under such conditions a person can at least be relieved of the companionship for longer or shorter periods of the day. If a hysterical person is made to live with a nurse she does not care

about, or even dislikes, she cannot escape from her and the results will be disastrous. If, then, the medical man desires to restore his patient to health he must take the greatest pains in selecting the nurse who is to take charge of the case, and he must, if necessary, discharge one after the other till he gets the right one, if the two are not found to be happy together. And no nurse ought to resent being relieved of such a charge for such reasons; no one gets on smoothly with everyone they chance to meet. Looking upon hysteria as mainly an abnormal condition of the brain of a functional kind, one would expect to be able to influence it mainly through healthy ideas and pleasurable emotions; and just as ideas and impressions received from another case may lead to the abnormal movements called chorea, so ideas and impressions derived from the association with healthy-minded, congenial companions may lead back to healthy conditions of brain action. By far the most important of all considerations in the treatment of hysteria is, then, the selection of those who are to be with the patient, and most of all, of the nurse who is to take charge. But the masseuse is almost of equal importance. Medical men are inclined to think that if they supply a skilful masseuse that is all that is required. But consider: the masseuse spends generally two or three hours a day with the patient, and her influence on the patient's mind may have as great an effect as her massage in favouring (or the reverse) a return to health. Personally I am in the habit of considering, as far as possible, the character of every one who comes into contact with hysterical patients, knowing full well that personal influence plays the most important part in their recovery. This is the natural and healthy form of "suggestion," and it is free from the dangers connected with that which is more technically called "suggestion."

As regards drugs one may say that they play a subordinate *rôle* in the treatment of the disease. Tonics, of course, help to restore the physical condition if it has suffered, but strychnine, one of the most active nerve tonics we possess, generally produces discomfort. Bromides are almost universally employed; and looking at the view we have

enunciated as regards the pathology of the disease one can well see how they can, by depressing the energy of the cortical centres, diminish the hyperæsthesia and irregular nerve discharges which occur. But the same effect will be produced upon those centres which are already unable to function normally, and will increase the sensory and motor paralyses, and diminish the controlling and energising nerve discharges which tend to a re-establishment of the lost equilibrium. Bromides, therefore, ought to be used in great moderation, as drugs which may relieve the discomforts of hysteria, but have a tendency in the long run to retard recovery.

Another disease of the nervous system of great importance is NEURASTHENIA. In some ways it resembles hysteria, and yet it is very different. Inheritance probably plays an important part in both; that is to say, not direct inheritance of the disease itself, but rather of a central nervous system which lacks certain characters which a perfectly sound one possesses. As we are in ignorance as to the actual pathology of either one or the other condition it is very difficult to describe what those characters are. In hysteria there is an absence of that steady, equable, well-balanced nerve action which is seen in healthy people. The consequences are explosive discharges on the one hand and sensory and motor paralyses on the other. In neurasthenia no such symptoms attain prominence; but a condition of simple exhaustion of nerve energy is the main characteristic.

Inheritance presents itself in the form of a tendency to the production of such exhaustion; an absence of staying power; an incapability of enduring unscathed the amount of stress and strain which a healthy nervous system can endure. Hence it is more common in men than in women; as the mental and physical strains which men have to bear are greater than those which befall the opposite sex. And hence, too, the proneness of the condition to occur in hysterical subjects, who possess a nervous system which is easily exhausted. Neurasthenia is supposed to be much more common than it used to be, and, looking upon it as an exhausted condition of the highest nerve centres from strain,

the conditions of modern life account for this. Its onset is generally gradual, and slowly produced by life's worries, anxieties, and overwork. But it may occur suddenly after physical injuries; though even in this case weeks may often elapse before it declares itself. In relation to this fact it is of interest to note that Obersteiner says it has been shown that injuries, such as blows on the head, may give rise to degeneration of nerve-fibres which extends a long way into the nervous system.

A sudden emotion may likewise produce profound neurasthenia. A patient of mine suffered from an absolutely incapacitating attack of the disease owing to hard work, exposure to sun in India, and other unfavourable conditions. He recovered very slowly, after a year of treatment, but so completely that he went through the South African campaign without difficulty and returned home well.

After an interval of six years from his former attack, during which he said he had been in better health than he had ever been, he returned to me physically incapable of going about and unable to do mental work, or even to read a book or a newspaper. He had stubborn insomnia, and great hyperæsthesia as regards sounds. He was, indeed, fit for nothing. This state of affairs had developed quite suddenly, owing to his being refused by a lady, whom he had known for a considerable time, and who had given him reason to believe that a proposal of marriage would certainly be accepted. The sudden emotional shock produced by the refusal resulted in a sudden and complete breakdown.

It has already been remarked that one is born hysterical, and in a similar sense it may be said that one is born neurasthenic. In both cases one is born with a nervous system which will show its defects under unfavourable circumstances. But, as has also been remarked, such unfavourable circumstances may never overtake the person; and so the weakness is never discovered. Neither fits, nor sensory and motor paralyses, nor contractions, occur as a rule, in neurasthenia. The most striking phenomena are mental and physical weakness; a condition in which fatigue easily follows muscular or mental exertion even of a very

moderate kind. The patient cannot, for example, apply himself to his business as he used to do; he cannot concentrate his attention; his memory is defective. Nor can he, owing to inertia, indulge in the recreations and exercises which before gave him pleasure. One of the most prominent symptoms is insomnia, which is not only a result of his disease, but also aggravates it. Restlessness, fretfulness, irritability of temper, hypersensitiveness, visual and auditory, discomforts about the head and upper part of the neck, especially posteriorly, may all be sources of great trouble. Things which never caused worry before do so now. This hypersensitiveness on the mental side has its counterpart on the physical. Pains and discomforts may arise in all parts of the body, suggesting the presence of disease and causing anxiety to the patient. The various organs of the body do not ordinarily in healthy conditions force themselves into notice. But the neurasthenic patient may be painfully conscious of his natural need of breathing: of the action of his heart; of the presence of food in his stomach; of the presence of small quantities of urine in the bladder or of fæces in the large bowel, and, indeed, of almost everything which is going on in his body, which the healthy man does not perceive, and these abnormal sensations are not only uncomfortable but are also disquieting, as suggestions of all kinds of diseases. He suffers from indigestion, flatulence, and a sinking in the epigastrium. A dread that something serious is going to happen may haunt him; and he may be troubled with agraphobia and claustrophia. The deep reflexes are exaggerated as a rule, and a very constant phenomenon is a feeble pulse of very low tension.

Neurasthenia is the result in most cases of prolonged adverse conditions, which make constant calls on the highest nerve-centres without due intervals of rest and relaxation being allowed for recuperation. Constant worry and strain of one kind or another are almost always the antecedents, though it is true that sudden emotion on trauma may have similar effects. But, as has been pointed out, even under the latter circumstances, the condition may not follow at

once, but after an interval of a few weeks. The disease is due to sheer exhaustion of the highest nervous centres, and this exhaustion is pretty certainly accompanied by alteration in the nerve-cells—in fact, by degeneration. In hysteria this can hardly be the case; for, if it were, the sudden disappearance of motor and sensory paralyses could not occur.

The persistence of neurasthenia, and the length of time which it takes to disappear, also speak in favour of a degenerative change having occurred in the nerve-cells, at least of the cerebral cortex. Indeed, I believe that many patients never succeed in regaining the condition they enjoyed before they broke down, but remain incapable of doing the work they used to do. Even at their best they can no longer work with rapidity, or stand worry. They have to reduce themselves to a slow, steady pace, and then, and only then, can they accomplish much.

Recent investigations have shown that anatomical alterations occur in nerve-cells when acting in an ordinary way as well as when pushed to fatigue. van Gehuchten sums up the results of these investigations as follows:—

The cellular protoplasm during normal activity undergoes swelling, which produces an increase in volume of the body of the cell and diminution in the quantity of the chromophile substance.

The nucleus likewise augments in volume.

When pushed to fatigue there is a diminution in the protoplasm of the cell, and the nucleus diminishes also, and undergoes an alteration in shape and modifications of its chromatic substance.

Hodge found similar alterations in the nerve-cells of fatigued animals, such as the pigeon and the honey-bee. These disappeared after a night's rest. In neurasthenia, as a rule, the antecedent stress and fatigue have been prolonged, and there has been but little opportunity of recuperation owing to the accompanying insomnia. Hence one would expect much more profound atrophy and other pathological changes in such cases.

As regards traumatic neurasthenia which follows severe

railway and other accidents, it can hardly be doubted that the nervous system, and especially the brain, is actually physically injured. In examining *post-mortem* cases which have succumbed to their injuries one often finds more or less widespread bruising of the surface of the brain. This must involve the neuraxons aud dendrites of many neurones. Recent researches have proved that not only section but many injuries of the neuraxon involve not only peripheral changes in it but alsc alterations in the nerve-cell from which it emanates. van Gehuchten gives the following: Alteration in the chromophile substance—chromolysis; swelling of the cell body; displacement of the nucleus to the periphery.

The chromolysis commences about forty hours after the injury, and lasts fifteen to twenty days. Then occurs a re-formation of the chromophile substance and a return to normal in about 100 days. The cells, however, may atrophy and disappear, the whole process being more or less rapid and complete according to the severity of the injury.

As regards afferent nerve-fibres it has been shown, at least in the case of those of the spinal ganglia, that when they are injured changes occur in the ganglion cells of a similar kind, only more rapidly, and destruction of the cell follows. It is probable, therefore, that injury of the protoplasmic processes or dendrites in nerve centres would entail changes in the cells with which they are connected. In a severe concussion of the brain, therefore, it may be assumed that such changes occur in a considerable number of neurones; and as one neurone transmits stimuli to others, the function of many uninjured neurones would probably be altered by a diminution in their normal afferent stimuli. Barker, in his book entitled "The Nervous System and its Constituent Neurones," 1900, says: "The importance of normal adequate stimuli for the welfare of the neurones in health can no longer be questioned. As van Gehuchten has emphasised, without stimuli there can be no life. For the maintenance of absolutely perfect function, the relation of stimuli to the reparative nutritive power of the cell must be absolutely and perfectly adjusted."

Sherrington has shown how division of the dorsal nerves of the cord produces a loss of tone in the cells of the anterior horn. In a delicate and complex organ like the brain it can easily be imagined, therefore, what changes in its function, as a whole, might be produced by such pathological alterations in certain territories, intimately connected as the neurones are with each other.

But it may be objected, how is it that many such cases get rapidly well after the action for damages which they have instituted has been settled, but not before?

The trial does not generally come on till a long time after the injury has been received, and during that period the damaged nervous system has been gradually returning to the normal, a process which experiment shows to be a lengthy one. But meanwhile other factors, such as mental depression with reference to the loss sustained by prolonged illness, anxiety about the future, including the question whether damages will be granted to the patient by the court, have been added, factors which of themselves might produce neurasthenia. When the trial is ended, and suitable damages have been obtained, the mental causes of the patient's neurasthenic condition are removed, and satisfaction and hope are free to exert their tonic influences on a nervous system which time and treatment have rendered capable of responding. To procure this result, it seems to me only natural that the railway company or other agency which has inflicted the original injuries should be made to pay, as they alone can administer the medicine which will cure the incapacity for which they are responsible.

To what extent spinal centres and the sympathetic system are involved in neurasthenia is as doubtful as in hysteria. The fatigue of body and mind which the victims of neurasthenia present can be quite accounted for by degenerative changes in the motor and other regions of the cortex, without assuming any changes in lower centres; and the fact that the deep reflexes are usually increased speaks in favour of cerebral exhaustion. It is pretty certain that, given sufficiently trying conditions of life, even the most healthy and resistant nervous systems may break

down and the patient become the victim of neurasthenia. Those who start life with a bad inheritance from the nerve side, and who have a tendency to develop this disease, may as regards quality of nerve-action be amongst the most brilliant members of our race; but it is in quantity, or staying power, that they are deficient.

Prolonged illnesses, and especially the specific fevers, may leave the patient in a neurasthenic condition; and of all of them perhaps influenza is the worst. Febrile diseases, due, as they generally are, to micro-organisms and their specific poisons, are well known to produce granular degeneration and other changes in the cells of the body; and it is highly probable that the neurasthenia they leave behind is due to such changes in the cells of the highest nerve centres.

Neurasthenia is one of the most seriously incapacitating conditions, and its long duration, even under the best treatment, makes it very trying both for the patient and his friends; and what makes it even worse is the view which the relations and others often take of the case. Seeing the patient without any tangible local disease, or notable derangement of functions, but full of complaints and professing incapacity to do many things for which they cannot see any reason, they label him as "hysterical," and think that he shirks work and duties, and even pleasures, in which he could quite well take his share. But the incapacity is very real and requires careful and prolonged treatment.

If, as is supposed, a real degeneration of nerve-cells has occurred in neurasthenia, the length of time which the disease lasts, the slowness of the progress towards recovery, and even the failure of the patient ever to regain the condition of nerve energy which he formerly possessed, are accounted for. The main factor in treatment must be rest, mental and physical; not idleness, however, except in extreme cases. In the latter, where exhaustion is profound, the patient ought to be put to bed and the nervous system rested. It is advisable, however, that the circulation and the condition of the muscular system should be maintained in a healthy, active condition, and this massage, with

the aid of electricity, can accomplish, without any effort on the part of the patient's nervous system. Isolation may, or may not, be advisable. In some cases mere talking with friends fatigues, and it is better for the patient to be alone, with nurses only to attend him, for a while. But isolation in neurasthenia is required for purposes of rest, and not, as in the case of hysteria, so much as a means of bringing fresh and healthy influences to bear upon the patient. Plenty of wholesome food, and sometimes a small quantity of alcoholic stimulants, should be given. Insomnia is a great trouble, and narcotics must, in many cases, be administered, in order to obtain a moderate amount of sleep. Chloral hydrate, chloralamide, trional, &c., may be given in sufficient quantities to produce a good night's rest two or three times a week, but not every night. After a few weeks of this treatment the patient may be allowed to get up for longer and longer periods, be taken out to drive, and then allowed to walk. But whatever he be allowed to do must not be enough to produce discomfort, which really means fatigue; both mental and physical work and pleasure must fall short of this. And, in my opinion, in all cases of neurasthenia this is the most important point in the management of the patient, namely, the prevention of his doing more than that which leaves him without fatigue, the amount he can perform will then slowly but gradually increase. It is easy to lay down this rule, but an exceedingly difficult thing to carry it out, owing to the long duration of treatment. A busy man, for instance, wants to go away for a few weeks and then return to full work. But this he cannot do. Indeed, it may be said that in bad cases of neurasthenia the patient must permanently alter the conditions of his life, reducing his hours of work, and increasing the time spent in recreation and exercise; because the very fact of his having broken down shows that he has done more than he individually is capable of doing with safety. Patients will not, however, easily admit that there are individual limitations of power and endurance, and take it for granted that what others can do they can. Drugs indirectly help the patient considerably, procuring sleep, aiding digestion, which is

often disordered, preventing constipation, and playing the part of general tonics. But drugs cannot be relied upon to do more than this, and an indispensable factor in recovery is time.

And now, gentlemen, I have come to the end of my address, and I thank you for giving me a patient hearing. To many, I have no doubt, who are accustomed to settle their questionings and doubts by experiment, and who are impatient of theories, it will have been rather wearisome. But I shall be content if it draws their attention to a class of diseases which will well repay their consideration and scientific investigation.

A CASE OF "SUBACUTE COMBINED SCLEROSIS," WITH PROFOUND ANÆMIA.[1]

BY JAMES TAYLOR, M.A., M.D., F.R.C.P.

I FIRST saw the patient whose case I am about to relate, through the kindness of Dr. J. G. Fletcher, on July 10 of last year (1903). She was a woman of 63, and was sent to me on account of weakness of the legs and difficulty and unsteadiness in walking, which had commenced in December of the previous year, and had become gradually worse. There was nothing of significance in her previous health, except that for two or three years she had not felt well and her friends had remarked on her pallor and sallowness. Her illness began with a feeling of weakness in the legs. To this weakness was gradually superadded unsteadiness, and she was noticed to lift her feet quite unnecessarily high in walking. There had also been some wasting of the limbs and a feeling of weakness in the arms.

Tingling and a sensation of deadness in the legs had been a marked symptom ever since the commencement of her illness. There had also been some shooting pains in the legs, and she had had difficulty in passing water and occasionally incontinence of urine.

On examination at that time it was seen that her skin was of a pale yellowish tinge. Her hands seemed to be a little awkward, and there was distinct unsteadiness in standing and a high-stepping ataxic gait in walking. I could make out no sensory impairment, but she complained of shooting pains in the legs and of a sense of constriction round the waist. Both knee jerks were very active. I could find no ankle clonus, and I have, unfortunately, no note of the plantar reflex. She was extremely hopeful

[1] Read at the Harveian Society of London.

and sanguine, much more so than her condition seemed to warrant.

I regarded the case then as one of sclerosis associated with anæmia, and I anticipated that she would become worse. Her progress was steadily downwards, and on September 14, at Dr. Fletcher's request, she was admitted to the hospital.

On admission she was found to be much worse than when I had seen her, and I am indebted to Dr. Gordon Holmes for the very careful notes of the case during the time she was in hospital. She was quite unable to walk, she was much emaciated and weaker and her upper limbs were much wasted, and rigid and tremulous in the performance of fine movements. With the arms extended and the eyes closed there were athetoid movements present in the fingers, similar to those met with in tabes. The lower limbs were much wasted, very rigid, and subject to occasional severe spasms. There was much numbness and tingling in all the limbs, a girdle sensation at the waist, and dull aching pain in the lumbar region. There was definite impairment of sensibility to touch, pain and temperature all over the lower limbs. The upper limit was not definite. Stereognostic sense and sense of position were lost in all limbs. The knee jerks were active, there was slight ankle clonus, and the plantars were of the extensor type. She was quite unable to stand.

She gradually became much worse. On September 23 it was noted that her lower limbs were now flaccid and the knee jerk was not to be obtained. There was also complete incontinence of urine. Her muscles wasted, she became weaker and unable to feed herself, and her mind wandered at night. The anæsthesia had extended up to the second rib. The blood showed no qualitative change and there never were any retinal hæmorrhages present. She gradually became quite helpless; a bed sore developed; she usually wandered at night, but during the day her mind was wonderfully clear; she still showed the same hopeful and sanguine temperament which I had noted on first seeing her. She gradually sank, and died on September 27.

Of the *post-mortem* appearances I need only refer to the change in the nervous system, as the other organs disclosed nothing more than one would expect to find in the organs of a patient so emaciated and anæmic as she was. And I wish to acknowledge my great indebtedness to Dr. Farquhar

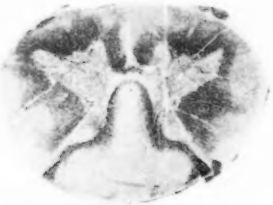

FIG. 1.

FIG. 2.

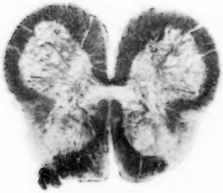

FIG. 3.

Sections in cervical, dorsal and lumbar regions of spinal cord.

Buzzard for the exceeding care he has given to this examination and for the excellent and full report with which he has furnished me. The brain showed no obvious abnormality. In the cord (figs. 1, 2, 3) there was extensive degeneration in the posterior columns, the only parts escaping being close to the gray matter. From the third lumbar segment downward

the sclerosis rapidly diminished. In the first sacral none was seen. The lateral columns were also sclerosed and here also the healthiest part was that next the gray matter; the crossed pyramidal, Gowers' tract and the direct cerebellar were all affected. The anterior columns showed less degeneration, except in the mid-dorsal where they were entirely degenerated, the only fibres escaping being those of the anterior limiting layer. Some degeneration was visible in the direct pyramidal tract as low as the fourth lumbar segment. The gray matter, so far as its myelinated fibres were concerned, did not seem to be altered. There was no degeneration in the posterior roots or in the anterior. The anterior horn cells were only slightly altered, apparently the result rather of malnutrition than of degeneration.

The cells in Clarke's column on the other hand had mostly undergone much change. They were nearly all shrunken, and many of them took no stain, being only represented by small masses of greenish pigment, retaining more or less the original shape of the cells. The cells of the posterior ganglia were also markedly pigmented and diffusely stained, rendering their details and outlines ill-defined.

The vessel walls did not appear pathologically altered, although they sometimes presented a hyaline appearance in sclerosed areas. The most noticeable condition was the small round-celled infiltration of the vessel walls, and of the surrounding lymph spaces. This was to be seen affecting many vessels in the posterior columns, and constituted a feature which is not common in cases of this kind. The membranes were unaltered, and the muscle fibre from the anterior tibial muscle showed no definite morbid change. The individual fibres were, perhaps, smaller than in a healthy subject.

In March, 1895, I read before the Medico-Chirurgical Society a paper on "Nervous Symptoms and Morbid Changes in the Spinal Cord in Certain Cases of Profound Anæmia." In this paper I described two cases in which the symptoms briefly were: gradual paraplegic weakness associated with impairment of the reflexes, with discolouration of the skin,

in one case so severe as to suggest at one stage Addison's disease, with sensory impairment, great anæmia and emaciation, and finally death. In those two cases there were found to be extensive degenerative changes in the spinal cord of the nature chiefly of sclerosis of the white matter in the posterior and lateral columns, and also in the anterior. A case exactly similar both in its clinical and pathological conditions had been published a few weeks before in BRAIN, by the late Dr. H. Bowman, and as this patient had been under my care as an out-patient at Queen Square, and as I had frequently seen her when she was in the hospital under that of Dr. Hughlings Jackson, I had the opportunity of comparing her clinical progress with that of the patients whose cases I published, one of whom was in hospital at the same time. Further, I had made the *post-mortem* examination of Dr. Bowman's case, and had prepared the sections of the spinal cord which he and I had examined and compared with those from the cords of my patients. The clinical histories in all these cases were very similar. In Dr. Bowman's case there was a history that her illness began with a severe flooding, and when she came first to see me as an out-patient suffering from intense headaches, she was in a profoundly anæmic state, and her appearance was so cachectic as to suggest malignant disease. She was admitted to hospital, and under the influence of rest, good food, and arsenic and iron, she improved very much and went out almost well. About six months later she returned, and by this time paraplegia had developed, and she rapidly became worse. In one of my cases there was a history that four years previously, in Canada, he had had a similar condition of weakness of the legs and ataxy from which he had made a complete recovery. In regard to the changes in the spinal cord in Dr. Bowman's case and my own, these were practically identical.

In none of these cases could the blood condition or the condition of the organs after death be described as characteristic of pernicious anæmia; but the cord changes found were obviously identical with those described by Lichtheim in 1887, as occurring in certain cases under his observation

of pernicious anæmia, and the clinical symptoms of paraplegia were similar. Minnich, his pupil, had also done some excellent work on this subject in the investigation of spinal cords from cases of pernicious anæmia in which paraplegic symptoms had not been present. In these he found scattered sclerotic foci, apparently the result of small hæmorrhages occurring in the cord, and also sclerosis in the posterior columns of varying intensity in different cords. He also found similar changes in the cords of patients who had suffered from other cachectic conditions, three cases of chronic jaundice, one of leukæmia, and one of tumour of the inferior vermis of the cerebellum. Lichtheim regarded the cord changes in his cases as the result of a toxic blood state, and Minnich's observations, while confirming such a view, suggested that some part of the sclerosis might be the result of hæmorrhagic foci in the cord, the hæmorrhages being similar to those occurring in profound anæmia, in the retinæ and elsewhere. It may be mentioned that in Dr. Bowman's case and in one of mine there had been retinal hæmorrhages observed before death.

During the last ten years numerous cases of a character similar to these have been published. Two important contributions came from Queen Square, continuing and supplementing the earlier observations of Dr. Bowman and myself, one by Dr. Risien Russell on three cases which he had observed, the other by the same writer associated with Dr. F. E. Batten and Dr. James Collier. In the latter paper, besides the observation of seven cases, there is a very comprehensive survey of the whole subject, and an interesting and suggestive attempt to divide the clinical history into three stages; the first being one of slight spastic paraplegia, with slight ataxy and marked subjective sensations in the lower limbs; the second, a stage of severe spastic paraplegia, with marked anæsthesia of the trunk and limbs; and the third, a stage of complete flaccid paraplegia, absent knee jerks, absolute anæsthesia, rapid wasting, paralysis of sphincters, and œdema of lower limbs. It may be said at once that there is no doubt that such a clinical history is more or less closely descriptive of

a large number of cases, as it is, *e.g.*, of that of which I have given the particulars at the commencement of this paper. It is not, however, applicable to all.

In my earlier paper, in 1898, I referred to an extremely important paper by Dr. James Putnam, of Boston—a paper which has not, I venture to think, received in this connection anything like the attention it merited. It was published in 1891, and described "a group of cases of system sclerosis of the spinal cord associated with diffuse collateral degeneration, occurring in enfeebled persons past middle life and especially in women." The symptoms generally consisted in progressive impairment of both sensory and motor functions of all four extremities, associated after a time with general wasting of muscles and emaciation. The fatal cases ran their course in about two years; in one case four years. Incoördination of movement was usually present. In several the knee-jerk was exaggerated and ankle clonus was present. In some obstinate diarrhœa was a prominent symptom. The anatomical changes found in the cord were extensive sclerosis of lateral and posterior columns and disintegration of the cells in the gray matter. Dr. Putnam also gave a short clinical account of four other cases with similar symptoms. In all there was great weakness with emaciation, and in some, at least, profound anæmia. In two the knee-jerk was absent.

In my earlier paper I expressed the opinion that notwithstanding some, as I consider, unimportant differences, these cases of Dr. Putnam's were to be regarded as belonging to the same group as Dr. Bowman's and my own, and also the cases described by Lichtheim and Minnich. I pointed out the clinical conditions in all as characterised by the gradual onset of symptoms, weakness and paraplegia, the association of these with, in all cases presumably, in some certainly, a greater or less degree of anæmia, in some profound anæmia, the variability of the reflexes, in some instances exaggerated, in others diminished or absent. And the actual changes found in the cords presented at least very striking resemblances—the same widespread sclerosis affecting the same parts of the cord and the same tendency to limitation of this change to similarly situated comparatively small areas in the

lumbar region. Dr. Putnam, however, had found changes in the gray matter which neither Dr. Bowman nor myself had detected in our cases. On the other hand, in many of the cases since described such changes have been distinct.

A somewhat futile discussion has taken place in reference to the question whether these changes are associated with *pernicious* anæmia.

There seems to be no definite agreement as to the clinical condition and state of the blood constituting this disease. But there seems to be little doubt that in some of these cases with cord changes, the condition has been one in which the blood changes supposed to be characteristic of pernicious anæmia have been present, and in which the organs after death have given the reactions supposed to be characteristic of this disease. But it is equally certain that all the cases with these cord changes have not been cases of pernicious anæmia. In the paper already alluded to I pointed out the association with profound anæmia, so profound as to be associated in Dr. Bowman's case and in one of mine with retinal hæmorrhages, and it must be acknowledged that in most of the cases anæmia has been a prominent feature. In one of Risien Russell's cases the condition of bad health had been apparently induced thirteen years before admission by a severe hæmorrhage occurring at a confinement. It is interesting to note that in Dr. Bowman's case the first symptoms had been induced also by a severe loss of blood. Yet although in the vast majority of recorded cases anæmia has been distinct, and in many cases profound, there have been cases in which anæmia was not so marked, but in those cases malnutrition was a distinct feature. Further, the cases are to be divided into two, it may be, somewhat indefinite groups, according to the duration of the clinical history. In some, and usually, I believe, those are the cases in which anæmia is marked, the course of the disease is rapid, and the fatal termination ensues in a few months from the onset. In others the progress of the disease is slower, and a year, or even several years may elapse before death ensues. Such a distinction was foreshadowed in my first paper, Dr. Bowman's and my own

cases falling into the first group, and Dr. Putnam's into the second. The distinction has been drawn more definitely in a monograph by Bastianelli, and it may be as he is inclined to believe, that anatomical as well as clinical differences distinguish the two groups. But it is scarcely correct to draw an anatomical distinction between the two in reference to the comparative affection of the lateral columns. Bastianelli says that in the first or rapidly fatal group the lateral columns are comparatively slightly affected; in the second or more chronic group the affection of the lateral columns is severe. The case I have just described disproves such a view, for it is not easy to imagine a more complete affection of the lateral columns than exists in this case in the dorsal region, and the clinical history of the case extended over only nine months. And Putnam points out that in one of his cases in which the symptoms extended over as long as thirteen years the spinal lesions were comparatively slight.

The question has also been raised whether these changes in the spinal cord are the result of vascular lesions. I have already expressed the view that, to a slight extent only, they probably are. In certain cases a hyaline degeneration of the vessel wall has been described, yet this has not been constant, and the changes have not been most marked in the vessels related to the most markedly sclerosed areas. Yet I think it possible that small hæmorrhages may occur from vessels in the cord as they do from retinal vessels, and give rise to small sclerotic foci, which coalesce with the larger areas which arise from some other causes. In one of the cases in Drs. Russell, Batten and Collier's paper there was a hæmorrhage into a posterior root ganglion, and in one of my cases there was a distinct unilateral difference between the sclerosis in the upper cervical and lower cervical segments. Yet it is only right to say that Russell does not share this view as to hæmorrhages accounting for any of the sclerosis.

In trying to account for this widespread sclerosis in these cases we seem to me to be driven back on the toxic theory, first promulgated by Lichtheim, and under such a view the sclerosis and the anæmia might both be the results

of the same toxin. What the supposed toxin is and where it is generated we do not know, but we do know of analogous conditions in which a chemical toxin does give rise to analogous cord changes. In diabetes, *e.g.*, we have a toxic blood state, and the observations of Williamson have firmly established the presence of sclerosis in the posterior column of the cord in many cases of diabetes. In pellagra also, a disease arising from eating unripe maize or even from drinking spirit made from such maize, we have changes in the cord strikingly similar both in extent and distribution to those present in the cases under notice. The symmetrical character of the sclerosis also is in favour of such a view, and the recent important work of Adami, Hunter and others is in favour of it.

One point remains to be considered, viz., the relation of the cases of ataxic paraplegia of Sir William Gowers to the cases mentioned above. Ataxic paraplegia is, of course, a clinical name, and it must be confessed that many of the cases above described are at some stage of their clinical history in a state of paraplegic weakness associated with ataxy. In Gowers' original cases the history was one spread over a considerable number of years as a rule; so is the history in many of Putnam's and Dana's cases, which one might call the American group. It is interesting that, in reference to a case published in BRAIN (vol. xiii., p. 356) by Dr. Michell Clarke of Bristol, as one of ataxic paraplegia, the author of that paper should himself have acknowledged to me after the publication of my first paper that he had gone into the matter again and re-examined his sections, and he had no doubt the case was one of the class I alluded to. Personally, I should be inclined to think that in this large class of cases, best known under the curiously hybrid name of "subacute combined sclerosis," there are very many varieties of the same disease, yet that the disease is essentially a unity, just as much as is tabes dorsalis, and that most, if not all, cases of ataxic paraplegia are cases of this disease. It is also a point of great interest that nearly all the cases described by American observers—Putnam, Dana and Taylor—should have run a comparatively long clinical

course. It is possible, however, that with the increased attention now devoted to this disease it may be found that they have the acute variety also in America, and that we have the chronic variety in this country more frequently than has been supposed.

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THE EFFECTS OF TOTAL TRANSVERSE LESION OF THE SPINAL CORD IN MAN.

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THE argument of this paper is that as the result of total transverse lesion of the spinal cord in man, not only are the knee-jerks and other deep reflexes permanently abolished in the region supplied by the caudal segment of the divided spinal cord, but in addition, the muscles waste and lose their faradic excitability, and the sphincters lose their tone, the only sign of self-action remaining in the isolated part of the spinal cord being the occasional presence of certain of the skin reflexes in much reduced degree. These phenomena occur in the absence of any recognisable structural change in the ventral horn cells, ventral roots and peripheral nerves of the paralysed region. That muscular wasting, loss of faradic excitability and loss of sphincter tone may occur in cases of transverse lesion of the cord was long ago pointed out by Sir William Gowers (10), but in his opinion the presence of these signs definitely proved the co-existence of concomitant or consecutive changes in the lumbo-sacral enlargement.

Attention is drawn to the fact that when the knee-jerks have disappeared in cases of progressive transverse lesions becoming total transverse lesions, for a certain time after such disappearance the knee-jerks may be again obtained subsequent to long-continued faradisation of the lower limbs. In other words, the lumbo-sacral centres in man have not sufficient self-action to maintain the muscular tone when severed from the higher centres, but they may be temporarily roused by peripheral stimulation into such a higher state of activity as will allow the knee-jerk to be

obtained. The importance of this fact, here recorded for the first time, in proving that the loss of knee-jerk is due to isolation from the higher centres and not to any concomitant injury to, or consecutive structural changes in, the lumbo-sacral centres is obvious.

These observations are based upon a considerable number of cases of complete and partial transverse lesions of the spinal cord which have been treated in the wards of the National Hospital, and my best thanks are here expressed to my colleagues upon the Staff of that hospital for permission to make use of the notes. I am indebted to Mr. Stansfield Collier for permission to observe and record two cases from St. Mary's Hospital.

The larger number of the cases were examined pathologically, while in several cases operative interference permitted the nature and extent of the lesion to be determined.

The cases were those of wounds of the spinal cord, fractured dislocations, compression paraplegia and acute transverse myelitis.

Brief reference is necessary in this place to the results of experimental transection of the spinal cord in animals and to the very wide difference of these from the effects of transverse lesions in the human subject.

In the cat and in the dog spastic paralysis results from transection, the deep reflexes are exaggerated and the most complicated reflex actions can be elicited, the "scratch" reflex, for example. Sometimes the transection is followed by temporary diminution or abolition of the deep reflexes, which lasts for a few minutes only.

In the higher apes transection of the cord is followed by spastic paralysis, but the spasticity is not nearly so marked as in the lower mammals. Complicated reflexes are not obtained. The knee-jerks are often absent for several days after the transection, but sometimes they return directly the effects of the anæsthetic has passed off. In these animals the autochthonicity of the isolated part of the spinal cord seems to be greater a few weeks after the transection than at later periods, and this capacity for self-action lessens as

time elapses. The term "isolation-alteration" has been applied to this slow process of physiological deterioration. The tendency towards temporary loss of the knee-jerk, its occasional permanent abolition, the spasticity little marked and the signs of self-action decreasing with lapse of time, make the effects of transection in the higher apes easily comparable with the effects of transverse lesion in man, the result being intermediate, as it were, between the spastic paralysis with exaggerated knee-jerks occurring in the dog and the flaccid paralysis with absent knee-jerks occurring in man.

I wish to draw particular attention to Professor Sherrington's description of isolation-alteration in monkeys (18) in connection with the loss of sphincter tone, muscular wasting, loss of faradic excitability and trophic changes, which gradually follow upon the occurrence of a total transverse lesion in the human subject. While such isolation-alteration is gradual with slow steps in the higher apes, it is gradual with rapid strides in man, and is manifest a few days after the lesion has become total.

In man it would appear that the disappearance of the knee-jerk is not synchronous with the occurrence of the lesion, since the knee-jerk has been repeatedly obtained in decapitated criminals (19), and it has been noticed to persist as long as ninety seconds after execution (20).

Since it was first pointed out by Dr. Bastian (4) that a total transverse lesion of the spinal cord entailed abolition of the deep reflexes, the effects of such lesions have been a matter of much controversy, the opinions of many authorities having been doubtless influenced by the effect of experimental transection in animals. At the present time it is almost universally accepted that a total transverse lesion results in flaccid paralysis, with loss of the deep reflexes, but it is also held that the knee-jerks may return after a lapse of time, and, further, several published cases of total transverse lesion in which the knee-jerks have persisted are accepted as undoubted facts.

As this paper deals only with the results of personal observations, I have considered it expedient to make but few

references to the extensive literature of the subject. It is allowable, however, to remark that writers upon this subject have not worked from the same standpoint as to what is a total transverse lesion, and, unfortunately, in some of the cases which have been published as examples of total lesion with persistent knee-jerks the totality of the lesion admits of doubt.

Pathologically, a transverse lesion of the spinal cord may be deemed total:—

(1) When the spinal cord is actually severed.

(2) When serial longitudinal sections through the region of the lesion stained by Weigert's method show no myelinated fibres passing through the length of the lesion, and when special methods for staining axis cylinders show a similar absence of these structures in the length of the lesion.

(3) When serial transverse sections through the region of the lesion, stained by a nuclear stain, show the whole area of transverse section to be necrotic.

The size of the spinal cord in the region of the lesion is no criterion of the extent of damage. Extraordinary reduction of the size may occur in pressure paraplegia with complete preservation of sensibility, as in Case 14.

Clinically, a physiological total transverse lesion may be considered to exist when there is absolute loss of sensibility, complete flaccid paralysis and loss of the deep reflexes.

Such a complete physiological abrogation of the functions of a transverse area of the spinal cord may exist without actual death of the elements involved, and these elements may recover more or less completely, as is frequently the case in pressure paraplegia and lesions involving evascularisation of a transverse area of the spinal cord.

When there is complete degeneration of all the elements of a transverse area of the spinal cord there can be little doubt that sufficient continuity for the maintenance of the spastic state and for the return of the knee-jerks may be re-established by the regeneration of conducting paths. That such regeneration does occur is strongly suggested by the conditions found in Cases 2 and 3, and by the behaviour of

some cases of acute myelitis, which begin to show marked improvement long after the onset. Immediate suture experiments upon the spinal cords of animals have shown that regeneration is possible in the spinal cord, and the researches of Bielchowski (5) argue strongly that it is no rare event as a sequel to lesions of the human nervous system.

In a large number of cases here considered the onset of symptoms has been gradual, a progressive lesion having at first produced partial, and later total, abrogation of the function of the involved part of the spinal cord. It is therefore of interest to consider the general order in which symptoms appear.

In another larger group of cases the onset has been sudden or rapid (fractured dislocation and acute myelitis), and the clinical picture of total physiological transection has been produced at once or early, but various degrees of recovery have subsequently occurred. The general order of regression of symptoms is therefore considered.

In a few cases the clinical picture of total transverse lesion has changed to one of incomplete lesion at a period so remote from the occurrence of the lesion and under such circumstances as to suggest strongly that the conductivity through the region of the lesion had been restored by the regeneration of the nerve-fibres.

With gradual lesions the order of appearance is practically constant as follows: motor paralysis and spasticity, equilibratory anæsthesia, sphincter paralysis, thermanæsthesia and analgesia, tactile anæsthesia, flaccidity with loss of the deep reflexes, progressive muscular wasting, progressive lowering of faradic excitability, progressive loss of sphincter tone. The motor paralysis and spasticity develop slowly and with even steps. Sphincter paralysis is variable, sometimes appearing long before any skin sensory loss is apparent, sometimes being delayed till skin sensory loss is marked. Loss of skin sensibility from increasing lesion, when once apparent, progresses much more rapidly than does the motor paralysis, and it would appear that sensibility to pain and temperature is always impaired earlier and to a greater extent than is sensibility to touch.

The place of appearance and the spread of sensory loss upon the limbs and body seem to follow one of two definite orders. Taking, for example, lesions of the lower half of the thoracic region: in the larger number of cases the sensory loss first involves the skin in the order of the length of the sensory neurones supplying it. The soles of the feet first become asentient, then the legs, and, later, the thighs; but when the anæsthesia reaches the trunk it extends upwards in segmental order.

In a smaller number of cases the sensory loss is first manifest over the most caudal segmental areas and extends upwards in segmental progression. It involves the segmental areas indirectly as their distance caudal of the lesion, the lowest sacral first, then the upper sacral, and, later, the lumbar areas.

The change from the spastic state to the flaccid state takes place rapidly in from twenty-four hours to a week, according to the acuity of the lesion, and is accompanied or shortly followed by loss of the deep reflexes. It never occurs till the sensory loss is complete over the lower extremities. Muscular wasting, lowering of faradic excitability and loss of sphincter tone commence with the onset of the flaccid state, and are progressive so long as the physiological transverse lesion remains total.

When there is recovery from the total physiological transverse lesion, the first sign of such recovery is the return of the knee-jerk and a gradual change from the flaccid to the spastic state. Sensibility returns in the order, touch, pain, temperature; and the lowest sacral segments usually become sentient before the others. The first signs of such return is often that the patient becomes conscious of the act of micturition, or of catheterisation, or of distension of the bladder during the routine lavage. In one case sensibility returned first in the feet.

Voluntary power returns on the flexors earlier than on the extensors, flexion of the toes being almost invariably the earliest voluntary movement possible.

In cases where recovery is complete the plantar reflex does not resume the flexor type till some time after the

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Voluntary power returns on the flexors earlier than on the extensors, flexion of the toes being almost invariably the earliest voluntary movement possible.

In cases where recovery is complete the plantar reflex does not resume the flexor type till some time after the

patient has completely recovered the use of the lower limbs, the extensor response being invariably the last of all spastic signs to disappear.

In the few cases where it is suggested that regeneration had taken place, the return of the knee-jerk, followed by a slow change from the flaccid to the spastic state, was the only constant feature. Further recovery was slight and its nature inconstant. In one case slight voluntary movement of toes of the left foot first returned, but there was no return of sensibility. In another case the patient became conscious of having his joints moved.

Sensibility.

The nature of distribution of sensory loss has been already discussed in the consideration of the onset of symptoms.

A noticeable feature of these cases has been the extreme rarity of any detectable zone of hyperæsthesia in the region immediately above the lesion. The writer has only met with it twice—once in connection with a cervical lesion where the third and fourth cervical root areas were distressingly sensitive (this case was not examined pathologically), and once in connection with a low thoracic total lesion where meningeal new growth was obviously involving several nerve roots above the level of the lesion of the spinal cord (Case 15).

Where pressure lesions exist the onset of complete anæsthesia does not mark an irrecoverable stage of the lesion, but the pressure must be relieved soon if recovery is to take place. Complete recovery occurred in Case 4, where a myxoma pressing upon the ninth dorsal segment was removed four days after the anæsthesia had become complete. Again, anæsthesia and complete motor paralysis are no proofs that a lesion is total unless flaccidity and loss of the deep reflexes co-exist.

Muscles.

Most important evidence as to whether a lesion is total or not is to be found in the condition of the muscles. When

the lesion is total the muscles become at once flaccid; they waste rapidly and their faradic excitability lessens, rapidly at first, then more slowly, and finally it may be lost. Where the lesion is not total slight wasting of the muscles occurs, but the tone is increased and the slight lowering of faradic excitability that may be met with seems to vary as the lesion occurs, rapidly or gradually. In a case of destruction of several of the upper thoracic segments by a wound, the muscles of the legs showed the faintest response to strong faradic stimuli on the fifth day after the injury. In the case of myxoma pressing on the ninth dorsal segment, the strongest obtainable faradic stimulus only caused a faint reaction on the third day after the onset of flaccidity.

The muscular wasting affects all the voluntary muscles supplied below the lesion equally, and, in the course of time, it may come to be as severe as that which occurs in cases of anterior poliomyelitis, with complete involvement of the lower extremities, and, in cases of destruction, of the cauda equina.

The lowering and ultimate loss of faradic excitability to stimuli applied through the skin is not accompanied by initial increase of galvanic excitability, nor by any marked increased reaction to "make" stimuli at the anode. It is important to bear in mind in this connection that an atrophied muscle always responds to faradic stimuli applied directly to the muscle, whatever be the cause of the muscular atrophy, provided that muscular tissue still exists.

If, for example, a pair of needle electrodes be introduced into muscles which have been for years completely paralysed as the result of anterior poliomyelitis or nerve section, a single faradic shock of moderate intensity produces a local contraction of the muscle in the region of either electrode, and thus contraction is readily detectable, owing to the lever movement of the electrode upon the point where it pierces the skin. This local contraction may be that of the intrafusal fibres of the muscle spindle, which do not atrophy as the result of section of the motor neurone supplying the muscle.

It has been argued that the muscular wasting and loss

of deep reflexes which occur in total transverse lesions of the spinal cord are the result either of coincident damage to the lower motor neurones of the lumbo sacral region of the cord, or of consecutive structural changes in these elements.

These explanations are directly negatived by the fact that the most thorough microscopic examination of the lower motor neurones in many of the cases failed to show the least departure from the normal in any part of their course, the richness of the tigroid substance and the distinctness of its arrangement, and of the cell processes, affording excellent demonstration of normal ventral horn cells in a state of rest.

Exception must be made in the case of the cells of Clarke's nucleus and the equivalent sacral nucleus, which invariably show marked signs of degeneration after transection of the cord. This fact was first pointed out by Schäfer (14), and it has been repeatedly confirmed by other writers.

Brasch has recently found degeneration both of the ventral horn cells and of the lower motor neurones in cases of total transverse lesions, with loss of the deep reflexes. He considers that this is the result of tertiary degeneration (isolation atrophy), and, in his opinion, the presence of such degeneration explains the flaccid paraplegia and loss of deep reflexes. The immediate onset of flaccid palsy and the immediate abolition of the knee-jerks, however, cannot possibly be the result of any such degeneration, nor can the rapid return of spasticity and of the deep reflexes, which occurs when pressure is relieved, after a physiological total lesion, be explained upon such grounds. If tertiary degeneration sufficient to cause flaccidity and abolition of the knee-jerks ever occurred, its clinical signs would be that after a total lesion the muscle tone and deep reflexes would gradually disappear, and this does not occur. Francotte, Leyden and Marinesco have also found changes in the ventral horn cells, Bischoff and Bartels have found the posterior roots of the lumbar region degenerated, while Oppenheim and Siemerling have found changes in the peripheral nerves of the lower extremities.

Slight abnormality of the ventral horn cells was met with in several cases where malignant disease, suppuration, or pyrexia had existed, but such changes were not greater than those which may occur in similar conditions apart from any spinal cord lesion.

Clinical evidence strongly supports the argument that muscular wasting, lowering of faradic excitability, and loss of deep reflexes under consideration, are the result of isolation of the lumbo-sacral region of the cord from the higher centres above. For in cases of complete recovery from a physiological transverse lesion, the writer has seen these phenomena develop rapidly and disappear with such rapidity after the removal of the cause of the lesion as to render any other explanation difficult. Further, in cases of acute transverse myelitis and fractured dislocation, where there is at first a total physiological lesion, these symptoms appear to disappear as soon as some conductivity through the lesion is regained.

Again, in those cases where it is submitted that regeneration had taken place, these signs disappeared. Most conclusive evidence in support of this argument is that for a few days after the occurrence of a total transverse lesion strong stimulation of the lower extremities often causes a temporary return of spasticity and of the knee-jerk.

The microscopic appearance of the wasted muscles is peculiar. The muscle fibres are greatly and regularly reduced in size (intrafusal fibres excepted). The transverse structure is ill-marked, but usually apparent; the nuclei are not increased. Fatty changes and vacuolation are not present, and there is only slight connective tissue proliferation and moderate periarterial thickening. These changes are strikingly different from those seen in muscles where the supplying neurone has been injured.

It may be remarked that Dr. Sherrington (17) has shown that certain muscles, notably those supplied by the third and sixth cranial nerves, show few histological changes as the result of section of their motor nerves, and that these muscles possess no spindles.

The Deep Reflexes.

A total transverse lesion of the spinal cord, whether physiological or anatomical, causes abolition of the deep reflexes below the lesion. The return of the deep reflexes at any subsequent period is significant that the lesion has not remained total, and that conductivity through the region of the lesion has again become possible either from recovery of elements of which the function has been in abeyance for some time and has been resumed, or from the regeneration of nerve fibres.

It would appear from the previously mentioned observations upon decapitated subjects that the deep reflexes may be retained for some short time after the occurrence of a total lesion.

The writer has had the opportunity of examining three cases of sudden transverse lesion within two hours of the occurrence, and has always found a condition of complete flaccidity with absent deep reflexes.

Where progressive lesion passes on to total lesion the writer has been able to demonstrate repeatedly that for some few days after the occurrence of the flaccid state, with loss of deep reflexes, strong and continued stimulation of the skin or nerves of the lower extremities has the effect of rousing the sleeping lumbo-sacral centres to such a degree of activity that their self-action is expressed by a temporary return of the deep reflexes and in some cases the presence of foot clonus.

The procedure was carried out as follows: A sterilised gilt needle electrode was passed deeply into the region of the external popliteal nerve below the head of the fibula. A similar needle was passed deeply into the calf. The two electrodes were connected with a strong interrupted current, which was allowed to act from five to twenty minutes, after which the conditions of the muscular tone and of the deep reflexes were tested.

It was found that if the state of flaccidity with absent deep reflexes had not existed more than three days the knee-jerks were easy to bring back. If this condition had lasted

longer than three days, as each day elapsed longer stimulation was required; the knee-jerks when obtained were more feeble, and they disappeared sooner. While after ten days prolonged stimulation was in no case followed by return.

The deep reflexes were obtainable for from five to fifteen minutes after the cessation of stimulation. Most marked immediately after the current ceased, they gradually became less, their elicitation lost its certainty and they disappeared.

Of special interest in this connection was the observation that in two cases of long-standing total transverse lesion, which are submitted as cases in which regeneration had taken place, and in which the knee-jerks returned late, the latter were first obtained after the patient had been subjected to faradism of the lower extremities in the way above described.

The Skin Reflexes.

These appear to be invariably abolished in severe transverse lesions with the exception of the plantar reflexes. The latter may be increased where the lesion is incomplete, where some recovery from a total physiological transverse lesion has occurred and when regeneration has taken place.

When the lesion is total, reflex action from the skin is much depressed, and it has been stated by several authorities that it disappears. It may be unobtainable directly after the occurrence of a sudden lesion, but the writer has found it present in three cases of sudden total lesion within two hours of the injury. The only reflex obtainable in the cases was a feeble response of the extensor type to stimulation of the sole of the foot.

Where life is prolonged with a total transverse lesion, it seems probable that all signs of reflex action slowly disappear.

Visceral Reflexes.

Priapism has been met with rarely in this series of cases, only in connection with partial lesions, and never where a total lesion existed. In the writer's opinion its presence signifies that the lesion is partial.

The condition of the sphincters in this series of cases did not accord with the classic description.

After the occurrence of a total lesion there may be temporary retention, and there may be reflex evacuation for a time, but the persistence of these phenomena seems to indicate that the lesion is not total. Constant dribbling with a small bladder has been the rule.

The anal and urethral reflexes have been repeatedly examined in these cases, and reflex evacuation tested by the application of cold to the skin supplied by the lower sacral segments, and it would appear that these reflexes and the sphincter tone are present directly after the occurrence of a total lesion, and that they gradually lessen till the orifices become patulous; reflex evacuation soon becomes difficult or impossible to bring about, active retention by the sphincters ceases and constant escape occurs, and the bladder slowly contracts and its lavage becomes a matter of difficulty.

The self-action of the centres of the bladder and rectum seems to be retained to a much greater extent in young children, and in this connection it is of great interest that in a case of a child of 6 weeks old, which has been published by Dr. Beevor, the writer had the opportunity of demonstrating spontaneous evacuation of the bladder, resembling both in periodicity and otherwise reflex evacuation, notwithstanding the fact that the whole spinal cord below the sixth cervical segment was destroyed and possessed no recognisable living nerve elements. A similar phenomenon has been recorded as occurring in very young animals after complete destruction of the lumbo-sacral enlargement.

Tympanitis.

Abdominal distension has been met with in a few cases where the lesion has involved the lower cervical and upper thoracic regions of the spinal cord.

It makes its appearance within a few days of the occurrence of the lesion, and if the patient lives it soon disappears. It seems never to occur as a late symptom of transverse

lesion of the cord, and in this series it has been present only in cases of fractured dislocation.

Œdema of the lower extremities was noticeable as an early and evanescent symptom in a few cases of severe or total lesion. It is prone to occur when malignant cachexia co-exists or when toxæmia and pyrexia are present.

Trophic changes have been very little in evidence in this series of cases, but slight changes in the skin and nails have been the rule where severe lesions have been long survived.

Bedsores do not occur if the skin is properly attended to. When present at the time of admission of the patients they have healed in many instances, where they had not reached an advanced stage.

Changes in the joints were not met with apart from rest adhesions.

Possible Clinical Utility of the Return of the Knee-Jerks after Faradisation as a Physical Sign.

The writer submits that in some cases of flaccid paraplegia the return of the knee-jerks subsequent to faradisation of the lower extremities may render it possible to determine whether there is implication of the lumbo-sacral enlargement and its nerve roots or not. For example, a case of progressive paraplegia from a lesion with its upper limit in the low thoracic region while under observation passes from the spastic to the flaccid state. If, soon after the onset of flaccidity and the loss of the knee-jerks, the latter return temporarily as the result of faradisation of the legs, it is certain that the lumbo-sacral enlargement and its roots are not involved in the lesion. But, if no such return can be brought about, it argues strongly that the lumbo-sacral enlargement or its nerve roots are so involved.

Case 15 is cited as a case in point, for many attempts were made to elicit the knee-jerks after faradisation, but without success, and the autopsy proved that the lumbar roots were seriously involved in the meningeal thickening.

It is of considerable interest that in the cases here

recorded complete recovery has followed the removal of pressure where the signs of total abrogation of function of a transverse region of the cord, namely, complete flaccid paraplegia, total loss of sensibility and loss of deep reflexes, have existed for as long as seven days. The recovery has been so rapid and so complete as to make it certain that pressure may produce complete physiological abrogation for many days without necessarily entailing degeneration of the nerve elements. The onset of the flaccid state in compression paraplegia is a sign which indicates that operative interference for the relief of pressure must be undertaken at once if it is to be of any avail, for the total physiological abrogation from compression signifies the presence of serious evascularisation, and the inevitable sequel of such ischemia —irreparable degeneration—cannot be long delayed.

Appendix of Cases.

Where several cases of the same clinical type have been examined, details of one only is given. Two cases similar to Case 3 have been examined, and many cases of similar nature to Cases 5, 6, 10, 11, 12 and 14 have been used in this communication.

Case 1.—*Destruction of several segments of the upper thoracic cord by injury.*

A man, aged 52 years, was admitted into St. Mary's Hospital under the care of Mr. Stansfield Collier in November, 1898. He had fallen from a considerable height, striking his back upon a rough projection. When examined one hour after the injury he had complete flaccid paralysis and complete anæsthesia below the fifth dorsal level. The knee-jerks were absent; a sluggish extensor plantar reflex was obtained on both sides.

The spinal cord was immediately explored. The posterior arches of several of the upper thoracic vertebræ were fractured, the fragments having been driven into the spinal canal. The spinal cord was found to be completely severed above the fifth dorsal segment, some two inches of its length being destroyed. Death occurred four days later. On the day of the injury there was retention of urine. On the following day the sphincter tone was present, but poor, and there was reflex evacuation. Sub-

sequently the sphincters were almost toneless, and dribbling occurred. There was slight tympanites. The condition of paraplegia remained the same till death, and the extensor plantar reflexes persisted. The muscles of the lower extremities wasted markedly, and on the day before death only a slight contraction could be obtained with a strong faradic current.

Remarks.—The totality of the lesion was demonstrated and the symptoms were classic. Of special interest were the rapid failure of the sphincter tone and the early lowering of faradic excitability.

Case 2.—Fractured dislocation, total lesion, probable restoration of conduction by regeneration. Autopsy.

G. M., aged 46 years, was admitted into the National Hospital under the care of Dr. Tooth on August 25, 1899. As the result of a fall twelve days before admission a fractured dislocation of the ninth dorsal vertebra had occurred.

There was complete flaccid paralysis and complete anæsthesia below the umbilicus. There was retention of urine, with occasional reflex evacuation of the bladder, for some weeks after admission; subsequently the sphincter tone became much reduced. The muscles of the legs were much wasted, and there was no response to faradism applied through the skin. Faradism applied by means of needle electrodes introduced into the muscles produced a slow local contraction.

The knee-jerks were absent; there was no foot clonus. There was a brisk plantar reflex of the extensor type.

Though this patient was repeatedly examined, the knee-jerks were never obtained until January 12, 1900, when five months after the accident the right knee-jerk was found present after the application of continuous strong faradism to the right leg. It persisted for about five minutes after the stimulation was discontinued, and then disappeared. The left knee-jerk was not obtained.

On the following day faradism was applied to both legs for about twenty minutes, after which both knee-jerks could be obtained, and they persisted for about fifteen minutes, right and left.

Two days subsequently, the knee-jerks being unobtainable, the experiment was repeated, and the knee-jerks appeared and persisted. Feeble at first, they gradually became brisk, and rigidity made its appearance in the legs. The reflex action of the lower extremities became exaggerated, but no sensibility

returned. A year later he was still completely anæsthetic below the umbilicus, but he could move the toes of both feet to a very slight extent. The knee-jerks remained brisk. He died of cerebral thrombosis on December 15, 1900.

The necropsy showed that the spinal cord was reduced to the size of a quill for a length of about 6 cm. in the lower part of the tenth dorsal segment. There was practically no bony deformity of the vertebral canal, the dislocation which caused the damage to the cord having presumably become immediately reduced.

Longitudinal sections through the region of injury stained by the Weigert-Pal method showed that there were a considerable number of fine fibres passing through the lesion, and that there was nerve continuity between the upper and lower limits of the lesion. The appearance and irregularity of these fibres suggested that they were fibres which had regenerated.

The persistence of signs of a total lesion of the spinal cord in the absence of any pressure upon the cord for five months, and the subsequent return of spasticity and slight voluntary power, suggest strongly that regeneration had taken place. This suggestion was rendered probable by the microscopic examination of the region of the lesion.

The return of the knee-jerks after prolonged peripheral stimulation is noteworthy.

Case 3.—*Fractured dislocation, total lesion, return of knee-jerks after faradisation six months later. ? Regeneration.*

J. R., aged 38 years, was admitted into St. Mary's Hospital under the care of Mr. Stansfield Collier in September, 1899. He had fallen a distance of ten feet, striking his head and left shoulder.

When admitted there was complete flaccid palsy and complete loss of sensibility to all forms of stimulation below the arms. The knee-jerks were absent. The plantar reflexes were extensor. Priapism and retention of urine occurred during the twenty-four hours following the accident. There was slight bilateral ptosis, and no light reaction could be obtained.

He remained in the same condition, except that the sphincter tone diminished markedly, and the muscles of the lower extremities wasted considerably and their faradic excitability became much reduced.

On March 25, 1900, after prolonged faradisation of the lower extremities, the right knee-jerks were obtained.

Faradisation was frequently applied, but the left knee-jerk was not obtained till three weeks later. Both knee-jerks persisted, and some degree of spasticity slowly returned. It is submitted that in this case the return of the knee-jerks was coincident with the restoration of some conduction through the lesion by regeneration.

Case 4.—*Pressure paraplegia. Total physiological lesion, knee-jerks obtained after faradisation for three days subsequent to the onset of the flaccid state. Operation. Recovery.*

G. P., aged 36 years, was admitted into the National Hospital under the care of Dr. Buzzard on October 13, 1898. Symptoms of pressure upon the spinal cord first appeared in July, 1898, and had become progressively more marked.

When admitted there was absolute analgesia and thermanæsthesia, with marked tactual impairment over the whole of the right lower extremity, and over the tenth, eleventh and twelfth dorsal and first lumbar areas of both sides. Over the left lower extremities there was marked loss to pain and temperature. There was spastic paralysis, and all movements of the lower extremities were possible, but were very feeble. There was slight dysuresis.

After admission the paralysis rapidly became absolute, and retention of urine occurred. Subsequently the anæsthesia became absolute below the ninth dorsal level. The knee-jerks gradually diminished, and foot clonus was no longer obtainable. On November 14 the paralysis became flaccid, the knee-jerks were not obtained on reinforcement, and complete incontinence of both sphincters set in, and the muscles of the lower extremities began to waste rapidly.

For the next four days the knee-jerks were obtained after strong faradisation of the legs.

As each day elapsed stronger stimulation was necessary for the elicitation; the knee-jerk, when elicited, was more feeble and lasted a shorter time after stimulation. On the fifth day the knee-jerks could not be elicited, even after thirty minutes' strong faradisation. Two days later the muscles reacted only to the strongest faradic stimuli applied to the skin.

On November 22 Sir Victor Horsley performed laminectomy, and removed an intra-thecal fibro-myxoma about the size of an acorn which was attached to the tenth left posterior root, and which was pressing into the posterior aspect of the cord.

The patient made an uninterrupted recovery. Five days after

the operation he could feel catheterisation, and the knee-jerks returned. Subsequently sensibility returned in segmental order from below up. The legs became markedly spastic. Voluntary power was first noticed on December 26, and subsequently it returned rapidly. On January 9, 1899, there was considerable loss of sense of position at all joints in the lower extremities notwithstanding that common sensibility had completely returned.

He was able to resume work as a farm steward in the summer of 1899.

Case 5.—Total lesion. Return of the knee-jerks, produced by peripheral stimulation, for a few days after the lesion had become complete.

A. S., aged 50 years, was admitted into the National Hospital on October 25, 1898, under the care of Dr. Buzzard, suffering with cancer of the dorsal vertebræ, secondary to a scirrhus of the left breast which had been removed. There was complete flaccid paraplegia and complete loss of sensibility below the tenth dorsal level. The knee-jerks were absent and the plantar reflexes were extensor.

There was complete incontinence, and the sphincter tone was only just present.

For six days after admission strong faradisation of the legs by means of needle electrodes was followed by a return of the knee-jerk, which persisted in diminishing intensity at the longest for five minutes.

Subsequently the anæsthesia extended as high as the seventh dorsal level. The faradic excitability of the muscles of the lower extremities became progressively lowered, and finally lost to stimuli applied to the skin. She died on January 10, 1899. The pathological examination was made by Dr. Risien Russell. The spinal cord was reduced to the size of a crow-quill in the seventh dorsal segment, and its substance was semi-diffluent at this point. There was complete evidence of total transection. The caudal enlargement and its nerve roots were normal.

Case 6.—Pressure paraplegia, total physiological lesion. Operation. Recovery.

C. G., aged 13 years, was admitted into the National Hospital under the care of Dr. Bastian on May 20, 1900.

His symptoms began six weeks before admission with severe pain in the left side, followed by weakness of the left leg.

On admission there was complete spastic paraplegia. There

was marked loss of pain and temperature below the sixth dorsal level. Very slight and indefinite tactile impairment over the legs. There was slight dysuresis. The knee-jerks were exaggerated, double foot clonus was present and the plantar reflexes were extensor.

On June 15 there was complete analgesia and thermanæsthesia in the paraplegic region, but tactile sensibility was almost perfect. On June 22 there was entire loss of control over the sphincters, with retention. Tactual anæsthesia was very marked. On June 30 the anæsthesia was complete to all forms of stimulation. The knee-jerks were absent, but faradisation of the lower extremities for five minutes caused them to reappear for a few minutes. The limbs were absolutely flaccid. Incontinence became complete. On July 6 Sir Victor Horsley removed a large number of hydatids which were compressing the spinal cord in the region of the sixth, seventh and eighth dorsal vertebræ.

On July 17 the knee-jerks were obtainable.

On August 2 heavy touches could be felt everywhere, and there was some return of sphincter control.

On August 16 the first sign of returning voluntary power was noticed, and thermanæsthesia and analgesia were nowhere absolute. He made uninterrupted recovery.

This case demonstrates the order of appearance and disappearance of the signs of compression. It exemplifies also the return of the knee-jerk after faradism when the lesion is physiologically complete.

Case 7.—Acute myelitis. Total lesion. Autopsy.

R. B., aged 56 years, was admitted into the National Hospital under the care of Dr. Risien Russell on October 9, 1901, suffering with acute syphilitic myelitis of forty-six days' duration. For ten days after the onset he had had retention requiring catheterisation, and occasional reflex incontinence; subsequently there was complete incontinence. There was complete flaccid paralysis and complete loss to all forms of sensibility below the eighth dorsal level. The right knee-jerks were never obtained; the left was obtained as a mere flicker for two days after his admission, and was not subsequently obtained. The plantar reflexes were extensor. There was extreme general muscular wasting of the lower extremities, and the muscles only responded feebly to strong faradism. There was almost complete loss of sphincter tone, and there was complete incontinence. Some œdema of the flanks and of the lower extremities was present. There were two

bedsores and severe cystitis. He died on October 18, 1901. The pathological examination showed that the lesion was limited to the ninth and tenth dorsal segments. The upper two-thirds of the ninth dorsal segment was entirely necrotic throughout the area of transverse section. No normal fibres nor axis cylinders could be traced through this part of the lesion. The caudal enlargement and its nerve roots were normal.

Case 8.—*Acute myelitis. Total lesion. Autopsy.*

E. C., aged 28 years, was admitted into the National Hospital under the care of Dr. James Taylor on August 12, 1902.

In June, 1892, he was seized with typical acute syphilitic myelitis. He had had retention of urine for a few days after the paralysis set in.

When admitted there was complete flaccid palsy and complete loss of sensibility below the eighth dorsal level. The knee-jerks were absent. The plantar reflexes were extensor in type. The sphincter tone was very feeble. There was complete incontinence. The muscles were much wasted, but responded feebly to faradism. There were marked trophic changes in the skin of the lower extremities; several bedsores and severe cystitis were present. He died on September 7, 1902.

The autopsy was performed six hours after death. The lesion involved the ninth and tenth dorsal segments. The upper half of the tenth dorsal segment was completely necrotic, none of the elements staining with alum hæmatoxylin. Sections stained by the Marchi method and by Weigert's method also showed that there was complete disorganisation throughout the area of transverse section.

The caudal enlargement and its nerve roots were uninvolved.

Case 9.—*Acute transverse myelitis. Total lesion. Clinical aspect after two years.*

J. C., aged 12 years, was admitted into the National Hospital under the care of Dr. Bastian on February 20, 1899, with complete paralysis and complete loss of sensibility in the lower part of the body and lower extremities. The family history was unimportant. He had always been delicate, and had suffered with measles, followed by a long lasting discharge from the right ear.

His illness began on January 1, 1897, when having gone to bed feeling quite well, he woke with severe pains in the back and in both arms. On attempting to get out of bed he was

unable to move his legs. In the course of the next day the legs and lower part of the trunk became completely paralysed, and he lost all sensibility below the level of the nipples, and he was unable to pass water. He remained in this condition till his admission. On admission he presented the following signs: The optic discs were ill-defined and woolly in appearance at their margins, as if a moderate degree of optic neuritis had been present at some time. The pupils were normal.

There was absolute anæsthesia to all forms of sensibility below the level of the upper border of the second rib. Sensibility was completely retained over the upper extremities.

There was complete flaccid paralysis below the second rib, except in the diaphragm, which acted well. No intercostal movement occurred, nor any movement of the abdominal muscles nor lower extremities.

The paralysed muscles were much wasted, and showed no response to strong faradism nor to strong galvanism applied to the skin. When needle electrodes were introduced into the muscles a feeble slow local response occurred with both forms of stimuli.

There was absolute incontinence of sphincters, with loss of reflex response.

The knee-jerks were absent, and no signs of reflex action could be obtained from the skin.

The legs were faradised with a strong current for an hour, but no reflex response could be afterwards obtained.

It is submitted that in this case a total transverse lesion of the spinal cord in the dorsal region had occurred, and that the absolute lack of any sign of reactive vitality below the lesion was the ultimate result of transection alone and not of concomitant lesion of the lumbo-sacral enlargement. For the clinical picture in this case was only so far more severe than in many of the cases of total lesion here cited as the longer tenure of life subsequent to the occurrence of the lesion might explain on the ground of progressive isolation—alteration. And in those cases in this series which have been examined pathologically, and which have approached closely to the above case in clinical aspect, no changes have been found in the lumbo-sacral enlargement of the cord which would account for its loss of function.

Case 10.—*Acute myelitis, recovering.*

A. E., a laundress, aged 20 years, was admitted into the National Hospital under the care of Sir William Gowers on

September 16, 1898, suffering with acute syphilitic transverse myelitis of six days' duration. There was complete flaccid paraplegia and absolute loss of sensibility below the tenth dorsal level. The knee-jerks were absent; the plantar reflexes were of extensor type. There was complete incontinence, and the anal sphincter was almost toneless. The muscles showed distinct lowering of faradic excitability. The knee-jerks returned on the sixteenth day of the illness, and some voluntary movement on the thirty-first day. Reflex incontinence returned before the knee-jerk, and some control of the sphincters was regained before voluntary power. Sensibility returned in segmental order, and at one period Brown Sequard's symptom was definite. She recovered sufficiently to work, but her gait is slow and spastic (1903).

This case is cited as illustrative of the clinical picture and course of many cases of acute myelitis in this series, details of which are omitted. It is a typical instance of the total physiological lesion, with its classic signs of flaccid palsy and absent deep reflexes, becoming by recovery a partial lesion, with its classic sign of spastic paralysis.

Case 11.—*Pressure paraplegia. Complete motor and sensory paralysis. Knee-jerks retained. Autopsy. Lesion not total.*

E. S., aged 36 years, was admitted into the National Hospital under Dr. Bastian on August 10, 1899. She had had the right knee excised for tubercular disease.

The symptoms of spinal tumour had appeared three months before admission, and had gradually progressed.

On admission there was complete spastic paralysis and complete loss of sensibility below the fifth dorsal level. There was reflex incontinence. The knee-jerks were exaggerated, and the plantar reflexes were extensor. She died on August 18 of general tuberculosis. The pathological examination was made by Dr. Batten. At the level of the fifth and sixth dorsal segments the spinal cord was much compressed and partly invaded by a tubercular growth. Sections of the compressed region stained by Pal's method showed a considerable number of healthy fibres passing through the lesion.

Case 12.—*An incomplete lesion, with absolute and persistent motor and sensory loss.*

A. J. C., aged 26 years, was admitted into the National Hospital under the care of Dr. Bastian on July 5, 1898.

He was seized in December, 1897, with a typical acute transverse myelitis affecting the lower thoracic cord.

There was absolute spastic palsy and absolute anæsthesia to all forms below the eleventh dorsal level. The knee-jerks were exaggerated, and first clonus was present. The plantars were extensor in type. The muscles were not markedly wasted. The sphincter tone was well marked, and there was reflex incontinence, and sometimes retention with dribbling. Priapism occurred till May, 1898, but not subsequently. He died on August 5, 1898.

The pathological examination was made by Dr. Risien Russell, who found that there was no evidence of complete transection, normal medullated fibres being found in numbers in the lateral columns throughout the length of the lesion.

This case demonstrates the fact that the persistence of complete paralysis and complete loss of sensibility cannot be taken as evidence that a lesion is total.

Case 13.—*Pressure paraplegia. Recovery after complete sensory and motor loss, which persisted many weeks. Autopsy.*

M. T., aged 19 years, was admitted at the National Hospital under the care of Dr. Buzzard on June 14, 1898. She had had an angular curvature for twelve years. The paraplegia has appeared five months before admission. There was absolute spastic palsy and absolute loss of all forms of sensibility below the twelfth dorsal level. There was marked relative loss as high as the ninth dorsal level, touch being the least impaired. There was reflex incontinence of the sphincters. The deep reflexes were much exaggerated. She stated that she had been unable to feel anything in the legs for several weeks. Laminectomy was performed by Sir Victor Horsley on July 5, and the pressure was relieved. She made an uninterrupted recovery, and was able to walk well on November 1.

In January, 1899, she developed acute pulmonary tuberculosis and died.

The pathological examination was performed by Dr. Risien Russell. The seat of the lesion was the ninth dorsal segment, where marked signs of old degeneration were visible, as was also evident in the ascending and descending tracts of the cord.

This case is recorded as demonstrating that complete spastic paraplegia and complete sensory loss of many weeks' duration are compatible with complete recovery of the functions of the spinal cord.

Case 14.— *Compression paraplegia. Great local reduction in size of the spinal cord without sensory loss and with some voluntary power. Autopsy.*

F. S., aged 27 years, admitted into the National Hospital under the care of Sir William Gowers on October 1, 1897, suffering with spinal caries. The paraplegia began in May, 1897.

When admitted there was complete spastic paraplegia. No sensory loss and no dysuresis. Treatment by rest and extension for nine weeks were not followed by improvement. In December, 1897, Sir Victor Horsley performed laminectomy. Fourteen days later some voluntary power returned in the toes. He steadily improved, and became able to walk with assistance. Subsequently extensive tubercular disease of the lungs became active, and he died on October 11, 1898.

At the autopsy, which was performed by Dr. Risien Russell, the spinal cord was found to be reduced to less than one-half its normal size for a distance of 7 mm. in the region of the sixth and seventh dorsal segments.

Case 15. — *Total lesion, with implication of lumbar roots. Autopsy.*

W. W., aged 88 years, was admitted into the National Hospital under the care of Sir William Gowers on January 9, 1900.

Symptoms of paraplegia had appeared five months before admission, and had progressed steadily. When admitted, slight voluntary movement of the left lower extremity was possible.

There was no power of movement in the right lower extremity. The legs were spastic. The left knee-jerk was exaggerated, the right was subnormal. There was loss of control over the sphincters.

There was marked analgesia and thermanæsthesia and less marked tactual anæsthesia below the ninth dorsal level. There was slight impairment of pain and temperature sense as high as the sixth dorsal level, while the fifth dorsal area was very markedly hyperalgesic.

On February 20 Sir Victor Horsley performed laminectomy, and found that extensive pachymeningitis was present.

On March 28 the lower extremities were quite flaccid, and the knee-jerks were unobtainable. Loss of sensibility was complete up to the level of the xiphisternum. Repeated attempts were made to elicit the knee-jerks after long-continued faradisation of the legs, but without success. Subsequently the sensory loss

spread to the arms, which were involved in atrophic palsy, and he died on March 22, 1901.

At the autopsy extensive hypertrophic pachymeningitis was found extending from the upper part of the cervical enlargement to the cauda equina. There was complete necrosis of the ninth and tenth thoracic segments, presumably from evascularisation. All the lumbar roots were involved in the meningeal growth, and they were all degenerate.

This case is cited as demonstrating the clinical value of the non-return of the knee-jerks upon faradisation of the lower limbs, shortly after the onset of the flaccid state. The sign indicated that the lumbo-sacral enlargement of its nerve roots was implicated, and that the flaccid paraplegia was not solely the result of the total transverse lesion, for in the latter cases the knee-jerks should have been elicitable after faradisation.

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ON THE PRIMARY STAINING OF THE RAT'S BRAIN BY METHYLENE BLUE.

BY JOHN TURNER, M.B.

One of the most curious and important features in neurohistology is the extraordinary affinity which methylene blue has for the elements of the nervous system, especially in regard to what Bethe terms its primary staining capacity, *i.e.*, the staining of fresh material, not previously acted on by chemicals, with basic dyes.

By different ways of applying the stain very different results are obtained; thus, at one time it picks out certain details and neglects all others, at another time these details fail to show and quite different ones are revealed.

Ehrlich was the first to apply the stain directly to the tissues, which he did by injecting it during life into an animal. In this case the great bulk of the cytoplasm of the pyramidal cells is scarcely coloured at all, but other cells, scattered irregularly about in the cortex, stain very darkly. There is shown also around some of the pyramidal cells a network of fine, beaded fibrils. My method, which gives a similar, but more detailed picture, enables us to obtain this reaction in dead tissues. This I have called a pseudo-vital reaction, and up to the present time have only succeeded in getting it occasionally, and, as a rule, in small isolated parts of the tissue.

Apáthy, Bethe, Dogiel and others, by a different procedure, succeed in staining the neurofibrillæ of the ganglion cells, but apparently their methods do not differentiate the darkly-stained cells above mentioned.

Under still different conditions the dye seizes on other details in the ganglion cells, and stains the so-called Nissl-bodies (which are probably structures not directly concerned

with nerve conduction), and leaves the rest of the cell body uncoloured. Bethe applies the term "primary staining" to tissues which have been dehydrated in alcohol or ether before staining, and therefore regards the colouring of the Nissl-bodies as primary, and Lenhossëk and I have shown that perfectly fresh tissues acted on only by the dye clearly show them.

As a rule, in primary staining by methylene blue, it is one or other of the nervous elements which are especially selected. The glia is largely neglected. However, during my attempts to get the pseudo-vital reaction in animals' brains, I have found that occasionally the dye specially picks out certain of the glia cells and structures which, I believe, are also derivatives from these cells.

I propose to give, in the following pages, a short account of these results, and also of some in which I have succeeded in obtaining the pseudo-vital reaction in the brain of the rat.

I shall deal first with the pseudo-vital reaction which I have been able to get in only one or two cases after keeping the dead animal for twenty-four hours in the oven, at 25° to 30° C., or else by taking out its brain and leaving this organ under similar conditions in a closed glass vessel. The staining mixture, consisting of 40 cc. of 1 per cent. methylene blue, to which 2 drops of lactic acid has been added, and 4 cc. of peroxide of hydrogen (10 vols.) was slightly warmed, and the brain put into it in a covered vessel, and left in the oven (25° to 30° C.) for six or seven days, then fixed for twelve to twenty-four hours in 10 per cent. molybdate of ammonium, washed, dehydrated, embedded in paraffin and cut. The successful results having been limited to very small areas in the cortex and striate body of two or three brains, I purposely give a very cursory account of the procedure, as I am still experimenting, and hope before long to be able to arrive at more constant and more widespread results. After a great number of trials it is evident (1) that the reaction does not occur in perfectly fresh tissue; (2) that the addition of a trace of lactic acid to the staining fluid somewhat facilitates matters (with this observation we must connect the fact that lactic acid is developed in

dead nervous matter); (3) any signs of putrefaction due to aerobic organisms prevent a successful result, and at the same time, by setting free ammonia compounds, give an alkaline reaction to the tissue.

During these investigations I found if the brain was subjected to a certain heat, when immersed in the staining fluid, that a very different result from that sought after was obtained. Scattered irregularly throughout the cortex and white matter were small, multi-branched cells which stained absolutely black, and which appeared to be glial. Furthermore the apices of the pyramidal cells frequently stained deeply and showed numerous fine, short, lateral projections, ending in knobs; and, lastly, around many of the nerve-cells beautiful Golgi nets were shown.

(*A*) Pseudo-Vital Reaction.

Cortex cerebri (median aspect adjacent to the corpus callosum).—The cytoplasm of the pyramidal cells (including the cells of the second layer) and the spindle-cell layer, stain a pale sky-blue and quite homogeneously; the nucleus is somewhat darker and densely granular; except the apex, the other protoplasmic processes are practically not shown. Scattered here and there throughout *all* layers of the cortex are small (an average size being 14 × 9 μ) multi-branched cells, not definitely orientated, staining nearly black and giving off extremely long, delicate processes, which spring abruptly from the cell body and pursue a very sinuous course. These processes are not regular in contour, but present a number of fine beads or more elongated thickenings, lying close together connected by finer strands, and with high magnification the thicker parts do not appear quite homogeneous, but have a loose structure, something like cotton wool, and in favourable cases they show numerous delicate, lateral twigs, similar to those on the processes of certain of the striate body cells. The processes ramify freely within the first layer (fig. 1). The pericellular beaded network is very scanty; indeed, scarcely more pronounced over the cells than in the intermediate regions. Its fibrils are

extremely fine and the black beads are generally about 2 μ apart, but the distance between them varies considerably as in human brains. So far I have not seen anything approaching the dense fibrillar felt work such as exists between the cells in the human cortex. The fibrils forming the pericellular net work appear very similar to the finest branches of the dark cells: in both cases the structures are beaded, but usually the dendrites appear less homogeneous and sharply cut than the fibrils of the network.

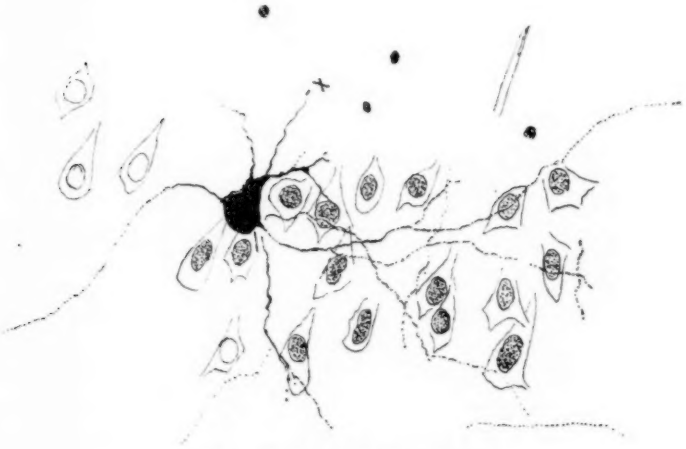

FIG. 1 (× about 500 diameter).

Small, darkly-stained cell in the second layer of the cortex. Its branches are extremely fine, and pursue a sinuous course for very long distances, retaining the same calibre. Note how they spring abruptly from the cell-body, and that some of them pass up into the first layer. X probably represents its axon passing up into the first layer.

Contrary to my universal experience with human brains, the first layer in rats, I find, takes on the reaction and shows considerable fine structural details. Fairly numerous extremely fine black fibrils run in it at all depths parallel to the surface. They have a clean-cut aspect and present at irregular intervals minute swellings, sometimes beads. Similar fibrils pass up into this layer from beneath, and

bend over to one side or the other. A fair number of nerve-cells are met with, staining the same light-blue colour as the cells in the lower layers. Occasionally also here and there the apices of pyramidal cells which have stained black are seen traversing this layer and bifurcating in it; sometimes they reach nearly to its outer margin, and it is to be noted that along their sides are closely clustered black dots, which join to the dark process by a delicate pedicle. In the few cases where I have been able to trace these dark apices to their parent cell the lateral offshoots were not apparent over the cell body, and only began to show at some little distance from it on the processes. These side twigs have very much the appearance of those on the dendrites of the striate body cells, but are shorter and more thickly clustered.

Cornu ammonis.—My remarks refer to the long pyramidal cells and the stratum radiatum. Only the bodies of the pyramids show, and they stain of an uniform pale blue, their nucleus not much darker. The ground work of the stratum radiatum and the spaces between the cells, where they are not so closely packed together, stain a dull pale gray, and coursing in the groundwork, usually in a direction more or less direct from the bodies of the pyramids to the granule layer, are a number of very fine, dark processes which originate from darkly-stained cells lying here and there among the pyramids or in their immediate neighbourhood. These dark cells are similar to those already described, but are, as a rule, somewhat larger (18 μ), and often more or less bipolar.

In the places where the bodies of the pyramids lie closely packed together they are marked off from each other by a darkly-stained beaded line, which, in places, gives off branches which pass over the cell bodies. Where the pyramidal cells do not lie quite so close together the fibrils course sparingly throughout the intermediate substance (see fig. 2).

Striate body.—This shows a structure very similar to the human caudate nucleus. Its cells are nowhere definitely orientated; the pale variety, much the more numerous, are chiefly indicated by their nuclei, as the cytoplasm is only faintly coloured just around these bodies. A few angular

cells stain deeply and give off comparatively short rectilinear processes, which dwindle rapidly and pass from view, and these I am inclined to regard as cells belonging to the pale

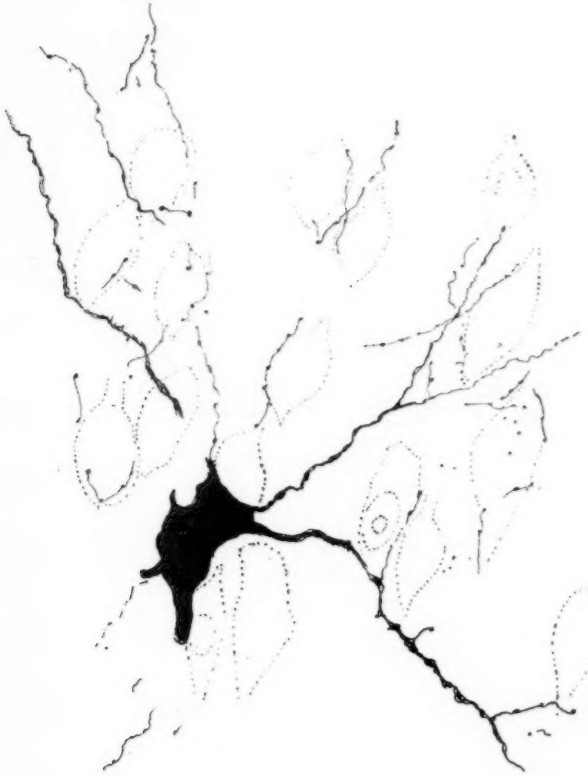

FIG. 2 (× 780)

Darkly-stained cell, lying in amongst the long pyramidal cells of the cornu ammonis (which are represented in outline only). Fine, beaded fibrils invest the pyramidal cells sparsely, and in some cases appear to take origin from the dendrites of the dark cell.

category, which have, for some unknown reason, coloured darkly.

The dark cells are exactly like those I described and figured in the human caudate nucleus (BRAIN, Autumn,

FIG. 3 (× 780).

A dark cell from the striate body. Delicate lateral twigs ending in a bead are given off from the more distal portions of the dendrites. At X a long, beaded fibril appears to arise from a dendrite.

1903). Their long, slender, sinuous processes form a coarse felt-work with each other, and generally in their distal parts

they give off numerous small, but distinct, lateral twigs ending in a knob a little distance from their origin, or else long fibrils with small heads on them at short intervals (fig. 3). Not all the dark cells show these "thorns"; the processes of some present a very irregular, moniliform, or coarsely-beaded appearance.

Numerous bundles of fine axons traverse this body on their way to the cortex, and where individual fibres can be detected they are of a pinkish hue and do not show definite beads, although their contour may not be perfectly smooth.

Remarks.

Although the results so far obtained are meagre, they show that the "dark" cells occur in both the first and second layers of the cortex and in the cornu ammonis, in which places I have not yet succeeded in showing them in the human brain. The presence in the first layer of the dendrites of these cells, especially of those cells which lie just below it, and which send up processes to ramify therein (*vide* fig. 1), might be supposed to militate against the idea that the beaded pericellular network is a derivative from these dendrites, because this layer is poor in cells. But in the new-born infant and in full-grown cats, as I have frequently observed, it does contain a considerable number of cells, the chromophilic elements of which are fairly well marked, and arranged in a similar manner to those in the pyramidal cells lying lower down in the cortex, a characteristic which at all events appears to mark them off as distinct from the dark cells. Very likely also in rats' brains these chromophilic cells are persistent throughout life, as in the cat.

I believe that not only do these dark cells form an intermediary system between the end afferent stations and the first efferent (*e.g.*, in the cortex), but that they are met with at afferent stations of different levels (*e.g.*, Clarke's column nuclei, gracile and cuneate nuclei, optic thalamus, &c.), interposed between the terminals of one level and the cells of the next; and that in all probability they will be met

with similarly disposed at the efferent stations of different levels, both cerebral and cerebellar.

Golgi long ago described cells to which he gave the name of intercalary; very probably these are included with others (notably Martinotti's cells) in my dark-cell system, and therefore I propose to call it the intercalary nerve-cell system.

Besides their staining peculiarities they have other well-marked characteristics by which they can be identified, and which, I submit, afford reasonable grounds for regarding them, wherever situated, as of like nature.

These are four in number, viz. :—

(1) *Dark staining.*—This, although a useful character by which their presence is readily revealed, is not alone to be depended on, for although, so far as I am aware, they always stain deeply, yet occasionally cells of the pyramidal system take on a dark colour also, and as this occurs very often in the brains of presumably healthy rats, especially in the cornu ammonis, it probably does not necessarily depend on a pathological condition, but possibly on some chemical alteration accompanying the functional activity of these cells.

(2) *Shape.*—Although in this respect they vary very much, yet they are extremely rarely pyramidal. They are usually square, pentagonal or oval, but whatever form they assume their protoplasmic processes spring from them abruptly and do not pass off by insensible degrees as, *e.g.*, the apical process of a pyramidal cell; sometimes, especially in the oval forms, a thick process springs from the cell in such a manner that one is unable to determine exactly where it leaves the cell body, but in such cases, after a very short course, it divides into two much smaller branches, which run for very long distances without alteration in calibre.

(3) *Dendrites.*—The way in which they arise has just been described. They run for very long distances without marked diminution in calibre, and are irregular in outline, with either moniliform swellings or lateral twigs. Their course is sinuous, and not, as is almost universally the case with dendrites of the pyramidal cells, rectilinear. This last feature I look upon as very characteristic.

(4) *Orientation.*—Unlike the pyramidal cells they are never definitely orientated.

In my previous communications I have given instances in the human brain which seem to demonstrate a direct connection between the protoplasmic processes of the intercalary cells and the beaded network around the pyramidal. In dealing, however, with such extremely minute structures, dogmatic statements must be received with caution; but, apart from such seemingly direct evidence, there is much of an indirect nature which also favours this assumption. I would especially call attention to the long, beaded fibrils which pass off from the dendrites of the intercalary cells of the striate body, and which have identically the same appearance as those forming the pericellular network. The objection that from this point of view the so-called law of dynamic polarisation is contravened, has with our present knowledge of the intimate structure of nerve-cells no longer much, if any, weight. The presence of neurofibrillæ, and the way in which they are arranged in the cells, shows that, as applied to the pyramidal cells, this doctrine is untenable. It is now a well-established fact, which anyone can verify for himself, that neurofibrillæ pass directly from one dendrite to another, and therefore, in whichever direction the nervous impulse traverses such a fibril, it must pass in opposite directions in the two dendrites in which a particular fibril lies. I am assuming that there are such phenomena as nervous impulses, and that they pass along neurofibrillæ. For the experiments of Bethe seem to show that the fibrils are the only material substance along which they can pass, for, according to him, they are the only structures which run continuously through the nodes of Ranvier.

(*B*) Heat-fixed Reaction.

If the staining mixture is added to the brain and cord and the whole subjected to heat in a covered glass capsule for some time, and then left in the oven at 25° to 30° C. for three or four days, one obtains occasionally, especially in young rats, a quite different reaction. I am unable to give any hard and fast rules, as my experience is not sufficiently extended,

but I have obtained successful results in several cases, more especially when the brain in its staining fluid was slowly raised to 60° C. on the water bath and then allowed to cool to 50° C. and again slowly raised to 70° C. and kept at this last temperature for some time, according to the size of the material to be stained (half to one or more hours). It is then placed in the warm oven (26° to 30° C.) for four days, and the subsequent treatment is as before described. In one case, which showed the lateral projections on the processes of the pyramidal cells, the brain had been subjected to a temperature of 100° C. for several minutes; in another, which showed Golgi nets around the Purkinje cells, the temperature was only raised to 50° C. and kept at this point for two hours. Under these circumstances, with successful preparations, one gets appearances quite distinct from the pseudo-vital reaction. No darkly-stained, intercalary cells, nor beaded network, nor other fine detail of an undoubted nervous nature is shown, but numerous glia cells are shown, sparsely scattered about in the cortex white matter, and in in the central nuclei and stem; and besides these well-developed cells with many branches, a number of round or oval bodies are seen, which vary in colour from a dull pink to almost black. They are about 9 μ in diameter and generally have a pale, round area within them occupying the greater part of their mass. These bodies are especially numerous in the white matter and amongst the cells of the nuclei of the stem. A variety of transitional forms between these just described and fully developed black glia cells occur; at one end of the scale there is an oval mass without processes; one passes from this to intermediate forms which show several short unbranched processes; at the other end of the scale the processes have become long, numerous, and freely branched, and the body less round (see fig 4). I have observed all forms of these cells in large numbers in the human cerebrum and cerebellum, treated in the manner first described for the rat's brain (*i.e.*, raised to 70° C.). In the latter region, cells which appear quite similar to the immature-looking forms in the rat's brain make up a large bulk of the entire granular layer (see

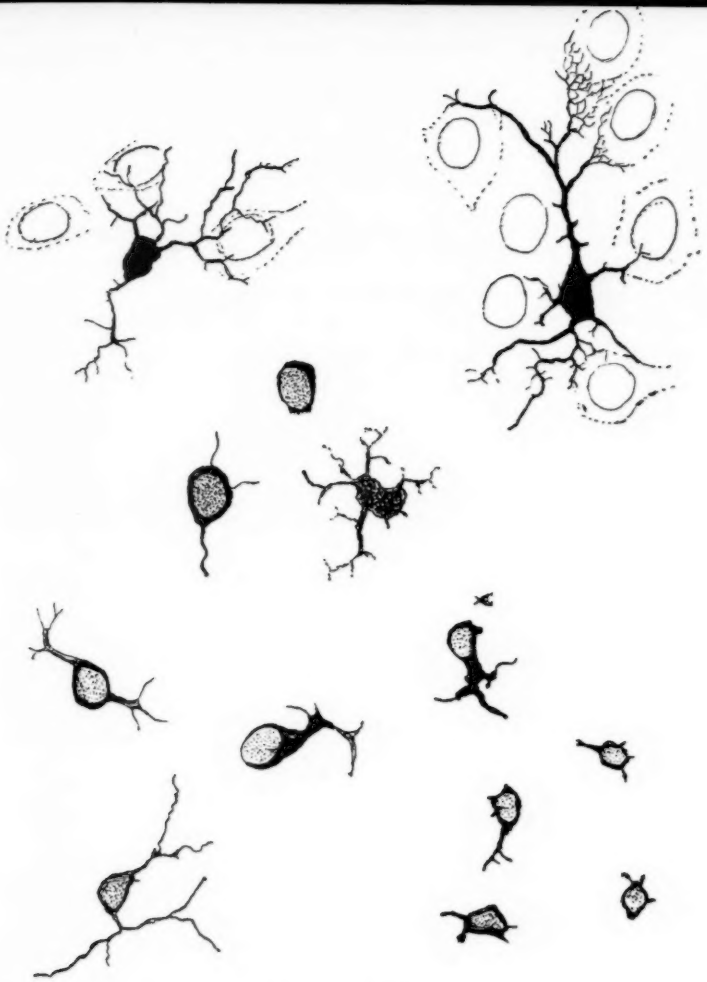

FIG. 4 (× 780).

In the upper part are two well-developed multi-branched glia cells lying in between the bodies of the long pyramids of the cornu ammonis. They are much smaller than the dark nerve-cells which are met with in this region (*vide* fig. 2). In the right-hand figure the end-branchings are shown (somewhat diagrammatically), breaking up into a lattice-work covering for the pyramidal cells.

In the middle of the page are three glia cells from the white matter, showing more or less immature-looking forms; the upper has no processes visible, the left-hand one has three stunted, unbranched processes, and the right-hand one has many. Note the pale area within two of these bodies.

In the lower part, to the left, are three well-developed glia cells from the molecular layer of human cerebellum. To the right are four small glia cells from the granular layer, with stunted branches. These are often clustered together into clumps of three or four. At **X** is a somewhat longer (8 by 5 μ) and more developed cell, which lies in the lowermost part of the molecular layer, and has the appearance of being a more developed form of the granular glia cell. (The pale centre, although represented as granular for facilities of reproduction, is really quite homogeneous).

right hand side of fig. 4, lower half). At the junction between this and the molecular layer and in the innermost part of the latter the cells become larger and more branched, whilst in the outer parts of the molecular layer well-developed forms occur (left hand fig. 4, lower half). In nearly all cases they show a pale central part.

My sections show very clearly that there is a very intimate relation between the branches of these cells and the trabeculæ of the Golgi nets; in fact, I believe they demonstrate a direct passage over of their branches into the net structure.

Golgi nets.—These are shown very distinctly, closely investing the bodies and dendrites of the cells of the dentate nucleus of the cerebellum, and most of the cells of the different nuclei within the pons, especially those of Deiters' nucleus, and the fore horn cells of the cord; they appear less distinctly shown around the pyramidal cells of the cerebrum, the cells of the corpora quadrigemina and the Purkinje cells.

They consist essentially of a close-fitting trellis structure, something similar in appearance to a piece of perforated zinc; the apertures are usually polygonal, and often contain a dark dot in the centre; the trabeculæ are slender. Bethe states that cells of different classes have nets of different patterns, so that from inspection of only a small piece of the net one is able to tell whether it belongs to a cell from the cortex, dentate nucleus, or trapezoid body, &c. The meshes over the dendrites are usually more elongated than those over the body. This authority also states that the net invests the axon for a short distance, ceasing either before or at the place where the myeline sheath begins.

It is very evident to the most casual inspection that this investment and the beaded pericellular network revealed by the pseudo-vital method are entirely distinct.

I have not been able to make out any extension of the trabeculæ of the Golgi nets to the matrix between the cells such as Bethe describes, but where two cells lie in apposition it passes continuously from one to the other, as he also notes. My preparations appear to show very conclusively that they

are derived from the terminal ramifications of the branches of glia cells, and although my personal acquaintance with these nets is confined to sections from several rats' and human brains, yet I have already met with, in the former, many instances which appear to establish this connection without doubt (see fig. 5).

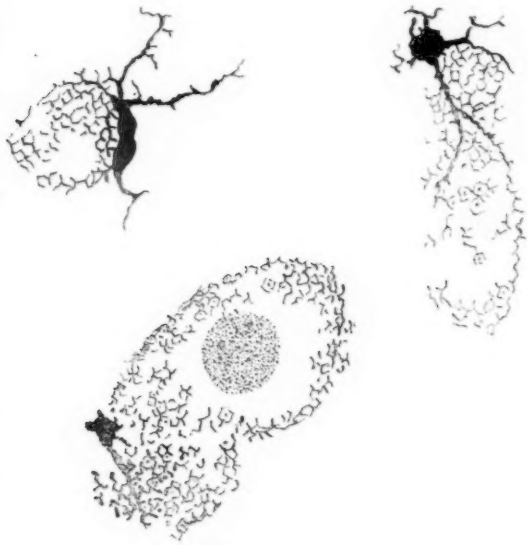

FIG. 5 (× 780).

Golgi nets surrounding, in the upper part of the figure, two cells from the dentate nucleus of the rat's cerebellum. In the lower part a large cell from one of the nuclei of the stem. In each case the darkly-stained glia cell from which the net appears to arise is shown. Note the pentagonal form of the mesh-work, and the frequent presence of a black spot in the centre. In the two upper figures the nerve-cell is merely indicated by its covering, but in the lower one the nucleus is shown.

Thorns.—In these heat-fixed preparations very often the apices and main dendrites of the pyramidal cells of the cortex stain somewhat darkly. The apices, under these circumstances, can be seen throughout almost the entire depth of the cortex, like a forest of pine trunks, lying closely together and ending apparently in the inner part of the first layer,

where they incline slightly to one side or the other; sometimes, especially in the case of the second layer cells, they bifurcate. The bundles formed by these apices are reinforced as they pass up by apices from shorter cells, so that they are more densely packed towards the surface than lower down. The cell body itself does not stain deeply, but the dark colour begins somewhat abruptly not far from it. The axons of these cells also stain deeply, but dwindle and disappear after a short course. In the more distal parts of the apical process, very fine, closely set, short twigs, ending in a knob, are seen passing away at right angles from its sides, and in cross sections through the dark apices these projections can be seen passing off all round like the spokes of a wheel (fig. 6). They appear quite similar to the lateral twigs already referred to, which show when the pyramidal apices stain darkly by the pseudo-vital method. Very often, also, in this view, the cut-through process is only dark on the surface and has an almost colourless centre. When examined under a high power it is seen that the dark colour is not uniform, but consists of darker granules on a somewhat lighter background. The beads can often be distinctly seen joined to the dark apex by delicate pedicles, but very often, in the immediate neighbourhood, others are seen scattered fairly thickly about and showing no connecting fibrils.

Remarks.

Golgi nets were originally described by the distinguished Italian histologist of that name in 1882. He ascribes to them an isolating function, and believes that they are a neurokeratin structure, but opposed to this idea is the fact, which Bethe points out, that this substance is not digested by pepsin, whilst the network is. Ramon y Cajal, Donaggio, Apàthy, and, recently, Held, regard them as glial in nature, and the two first as representing merely a peripheral extension continuous with the endocellular network of Golgi. Bethe and Semi Meyer believe that they are nervous structures, and Bethe represents them as extensions of axis

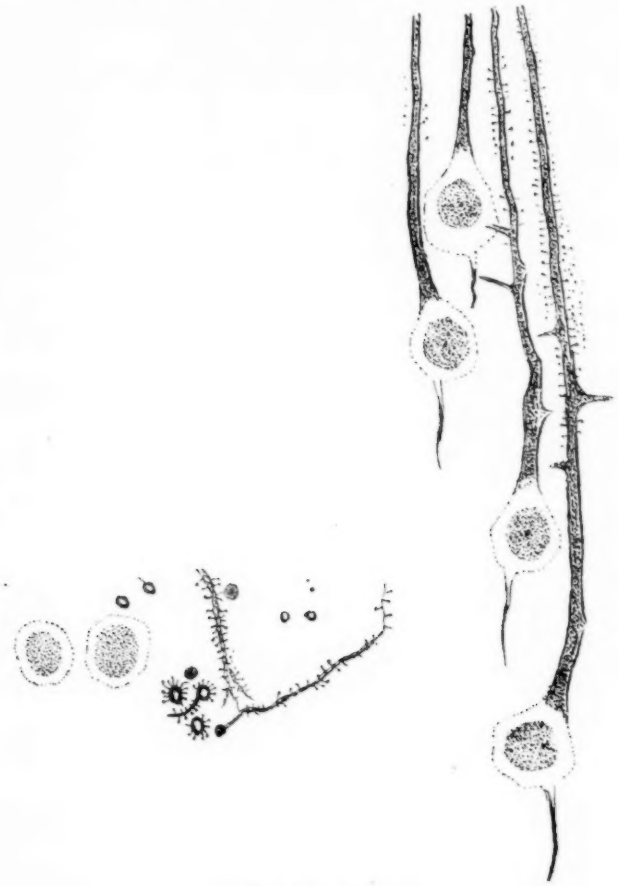

FIG. 6 (× 780).

On the right side are four pyramidal cells with darkly-stained apices. At some distance from the cell body fine dots are visible at the side of these processes, and generally a fibril connects the dot to the edge of the process. But, in places, collections of these dots occur which do not seem to have any connection to the pyramidal cells, as shown on the right side of the longest cell.

The left-hand figure gives a cross section of the pyramidal cells.

The apices now appear as a black spot or a black ring with colourless centre, and the little lateral projections come off all round like the spokes of a wheel. The long processes with thorns are dendrites. The two large, pale nuclei, with clear rims, represent two pyramidal cells cut through their bodies. Although these figures were outlined by aid of Zeiss's Camera Lucida, the lateral projections are so minute that it is not pretended that they have been individually traced, but only give the general appearance produced to the eye.

cylinder processes which, coming from other parts, join to the network and are thus brought into relation with the enclosed nerve-cell. The neurofibrillæ contained in the axis cylinders pass along in the trabeculæ of the nets, and leave them to enter the substance of the ganglion cells within. He believes that these nets, and a diffuse network which in places he sees in connection with them lying in the general ground substance, are identical with Nissl's hypothetical "gray" (but on this particular point Nissl does not agree with him).

My observations point strongly to the glial origin of these structures. The fact that I have only succeeded in showing them in preparations in which the stain only selected elements which appeared to be of a glial nature favours this view, but apart from such indirect evidence I have, as already mentioned, frequently seen what has every appearance of being a direct continuity between the branches of the glia cells and the network.

It is a pity that there are not greater facilities whereby different workers could compare one another's sections; if this were feasible probably many discrepancies and seemingly antagonistic views would be brought into harmony. This is the more to be desired as modern methods of staining nervous structures tend to become more special and limited in their range, so that taking any one method, whilst some details are shown, others which, perhaps, may be very closely related, are not revealed at all; and although one worker may not, and usually does not, limit himself to a single method, he will probably not reach an equal degree of perfection at all; thus, whilst he may obtain results which, after a careful examination of very many sections, appear to him convincing, he has not the time, nor perhaps the opportunities, to examine with equal thoroughness large numbers of preparations stained by different methods. Taking the case of these Golgi nets, I feel very strongly that my sections do show that they result from the splitting up of the glia cell branches, but I am unable to speak of their relation to the endocellular network, because the method I use does not show the slightest trace of these

structures. If, as Cajal and Donaggio think, they are both parts of one and the same thing, and in view of Holmgren's observations on the intracellular networks and "sap" canals in posterior spinal ganglia and gland cells, then they are probably a mechanism concerned with the metabolism of the cell.

On the other hand, the junction of axons with Golgi nets is strongly insisted on by Semi Meyer, and Nissl, after an inspection of Bethe's specimens, was also convinced of it. Nevertheless, in spite of the weight which opinions from such eminent observers carry, the minuteness of the structures under discussion must make it extremely difficult for anyone to definitely decide between actual continuity or mere proximity. It may be that the axons surround the ganglion cells, enter at the small pentagonal openings of the net, and break up within into fibrillæ. But, even should Bethe's observation prove correct, I should still be inclined to regard the network as not essentially a nervous structure, but as affording a covering for the fibrillæ, which Bethe states he has seen *within* its trabeculæ, without being itself concerned directly with the functional nervous activity of the ganglion cell.

On the assumption that the Golgi net is an extension of the axis cylinders rests the main fabric of Bethe's argument for the continuity throughout the central nervous system of individual neurofibrillæ as expounded by him in his recent publication, "Allgemeine Anatomie und Physiologie des Nervensystems," and this, to my way of thinking, is a weak link in his chain of reasoning. I myself believe in the continuity of nerve-cells with each other by their processes, but I think it occurs in a different way to that which he describes.

Thorns.—The thickly-clustered, delicate, lateral offshoots from the darkly-stained apices and dendrites of the pyramidal cells are to be seen, as already mentioned, not only in heat-fixed preparations, which do not, apparently, lend themselves to the staining of fine nerve details, but occasionally on the pyramidal apices which have, contrary to their usual custom, stained darkly in preparations which

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give the pseudo-vital reaction. Although they show a great resemblance to the projections on the dendrites of some of the darkly-stained cells in the striate body, I am inclined to think there is a distinction between them and those on the pyramidal processes, and that the latter are not integral parts of the cells to which they adhere. They appear to me to be finer, more closely clustered and shorter than those on the striate body cells. Without accepting the interpretation which Bethe gives to the function of the Golgi nets, perhaps, as he suggests, these lateral projections represent the stained trabeculæ of Golgi nets passing from the cellular to the diffuse portions of these structures. An objection to this view is that whilst we know that the Golgi net envelops not only the processes but the cell body, this latter part does not show the lateral projections, either with the pseudo-vital or heat-fixed methods. Speaking now of the appearances seen in heat-fixed preparations, if the Golgi net over the apices causes them to stain deeply, we may reasonably ask why the cell body is not also similarly affected. This may possibiy be due to a difference in the size of the meshes over the cell and processes; if those over the dendrites are smaller they might hold the stain so as to cause the whole surface to appear dark, whilst the bigger meshes over the cell body would fail to do so.

However this may be, I do not feel qualified at present to express any decided opinion as to their precise nature, except to reiterate what I have before stated, that they do not appear to me (especially in the heat-fixed preparations) to be integral parts of the processes themselves.

A few words in conclusion concerning the differentiation by the heat-fixed method of the granules of the human cerebellum. There is still much uncertainty about the nature of these bodies; true it is that Golgi's method shows here and there in the granular layer a small cell with ascending axon and claw-like dendrites, but these only occur in comparatively few numbers, and I have always felt that their presence was no sufficient justification for looking on all the granules as of the same nature. My preparations show that a very large number stain differently from the rest. These

stain deeply, show a few stunted processes and a pale central portion. They resemble very closely the immature-looking glia bodies in the rat's brain, and show grades differing only slightly one from another between the most immature form and the well-developed glia cell, and to my mind can with very little doubt be classified as glia elements. But as I have, up to the present, only seen these bodies in more or less pathological brains, I merely wish to draw attention to the fact without drawing any conclusions therefrom.

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All the figures were drawn by the aid of Zeiss's Camera Lucida.

NOTE ON A CASE OF DEFECTIVE DEVELOPMENT OF THE LATERAL CEREBELLAR LOBES IN A DOG.

BY THOMAS LEWIS.

(*From the Laboratory of Chemical Pathology of University College, London.*)

THE interest attached to the present case of cerebellar defect lies in the fact that it affords suggestions concerning the physiology of "circus movement" in an animal in whom the cerebral hemispheres were normal. Circus movement (*mouvement de manége*) occurs most commonly in animals in cases of lesions of the cerebral hemispheres producing hemianopia.

Luciani[1] has described the path taken by a dog in walking, after unilateral ablation of the cerebellum, as festooned to the opposite side, while the course followed by the same animal in swimming is stated to have been curved to the *same* side as the lesion.

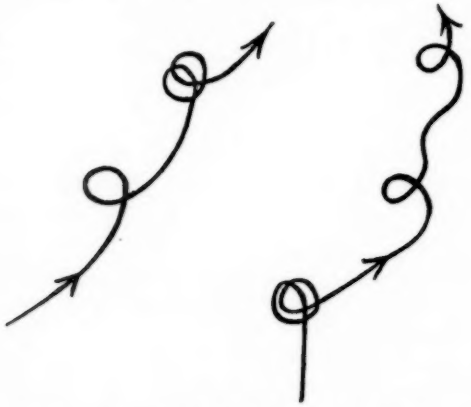
In regard to the ponto-spino-cerebellar mechanism there are at present generally recognised two main groups of movements, the first included under the term *mouvement de manége*, the second comprising rotatory movements around the longitudinal or transverse axis. In the case at present under consideration *mouvement de manége* was alone present, the diameter of the circle of movement being little more than the length of the longitudinal axis of the animal. Further, it is to be noted that in thus speaking of circus movement and in attributing it to a defect in the cerebellum, it is not suggested that it is strictly comparable to the large circular movements which Prof. F. O. Guldberg[2] has shown to be phylogenetically so important,

[1] *Il Cervelletto.* "Nuovi studi di Fisiologia normale e patologica," 1891.
[2] *Zeitschrift für Biologie*, T. XXXV.

and (in conjunction with Prof. C. Guldberg[1]) proved to be associated with asymmetrical development of the two halves of the body.

History of the Dog.

The dog, a collie, was described by its former owner as probably between 6 and 7 years old. It had been in his possession for four years; during the whole of this time the animal had rendered itself conspicuous by its curious gait and excitable disposition. It was in the habit of prowling up and down the road, executing at more or less short intervals complete revolutions to the left. The



movements were more frequent and rapid at such times as were likely to excite the animal—for instance, at the passing of vehicles and strangers. The condition was said to have become slightly exaggerated during the three months previous to its having come under observation. The state of the dog and its habits were watched for several weeks prior to its death, and the reported nature of the movements confirmed. The circles described by the dog were of very constant diameter, being between 1 and 1½ yards. Two tracings of typical paths taken by the dog in trotting show the frequency and direction of the

[1] "Etudes sur la dyssymetrie morphologique," Christiana, 1897.

circus movement during excitement. The dog's posture in lying, while under observation, was invariably on the side, with the trunk bent so that it was concave to the left. When the animal was unexcited progression was practically normal. Examination of the senses of sight and hearing showed no defect in the function of eye or ear. The reflexes in the lower limbs were equal on the two sides and normal.

These clinical observations being verified it was decided to test the corresponding cerebral mechanism by electrical stimulation of the cortex. The animal was placed under an anæsthetic and the following experiment was performed by Sir V. Horsley. The rectus femoris on each side was attached to a spring myograph, and the leg area on each side stimulated alternately. No irregularity of response was detected between the two sides. Further, excitation of the lateral lobes of the cerebellum was also without appreciable effect on the spinal reflexes. The animal was then killed and the brain and cord subsequently removed and examined.

Macroscopic Anatomy.

Cerebrum.—The hemisphere appeared to be perfectly normal and symmetrical.

Cerebellum.—The chief features of interest were:—(1) a general diminution in size relative to the cerebrum; (2) an increased width of the paramedian sulci, and greater depth of the fossæ on either side of the vermis; (3) a general shrunken aspect of the dorsal surfaces of the lateral lobes.

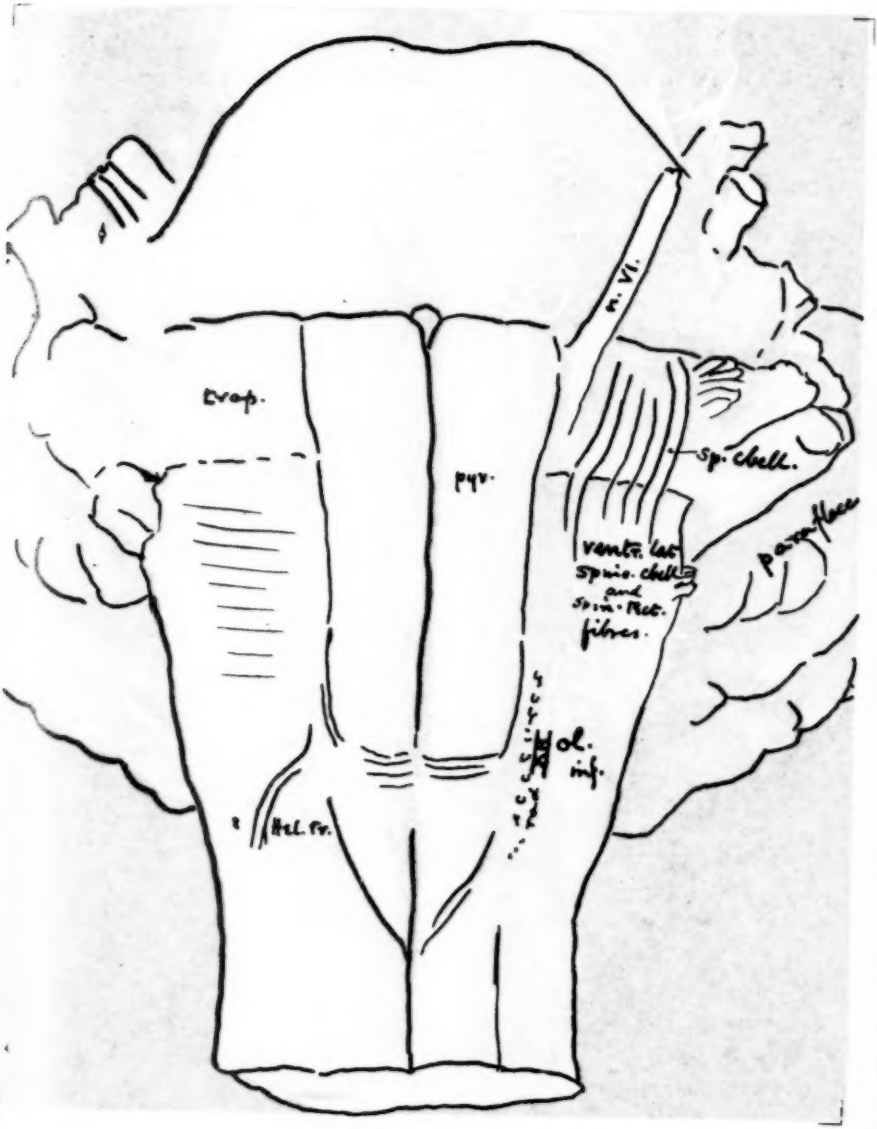
Pons and Medulla.

The basal aspect of the cerebellar tracts is shown in fig. 1, to which reference should be made.

Superior peduncles.—These tracts showed a slight general diminution in size.

Middle peduncles.—These showed no variation from the normal.

Inferior peduncles.—The *dorsal* cerebello-spinal tract, as it turned up to form the superficial layer of fibres of the resti-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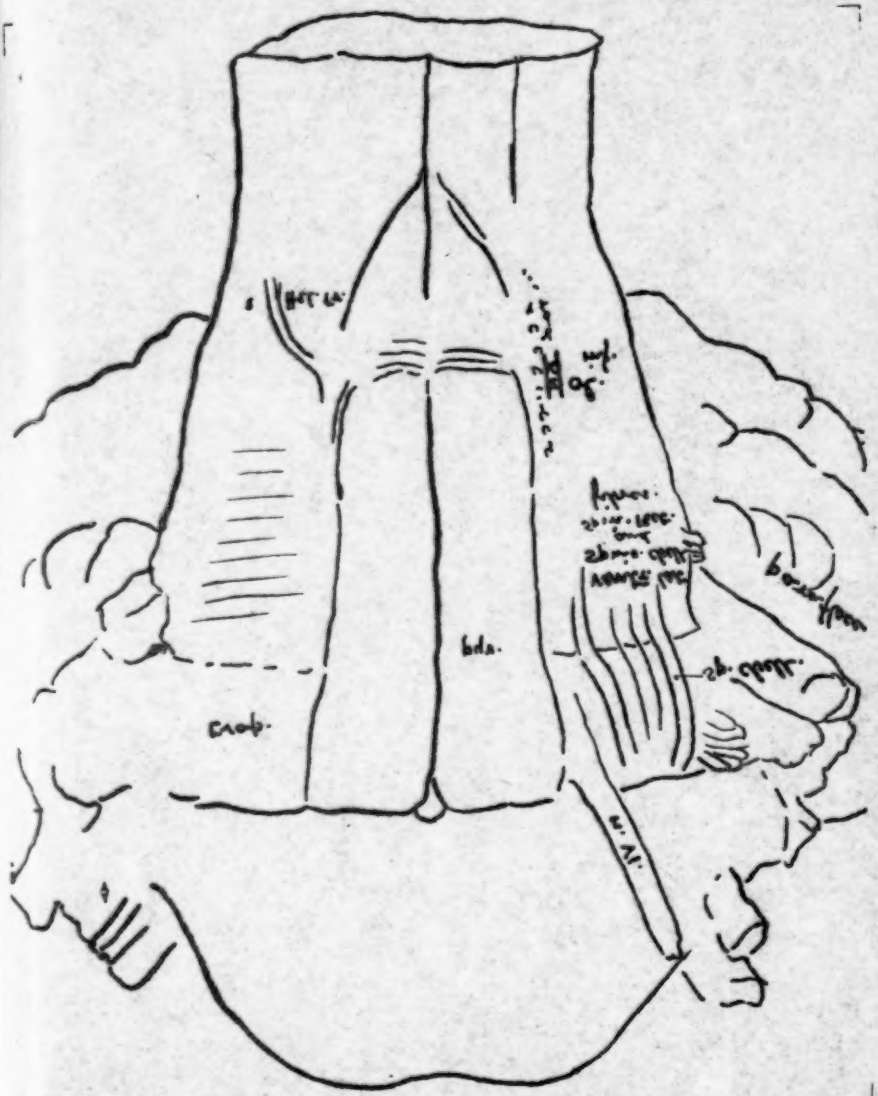


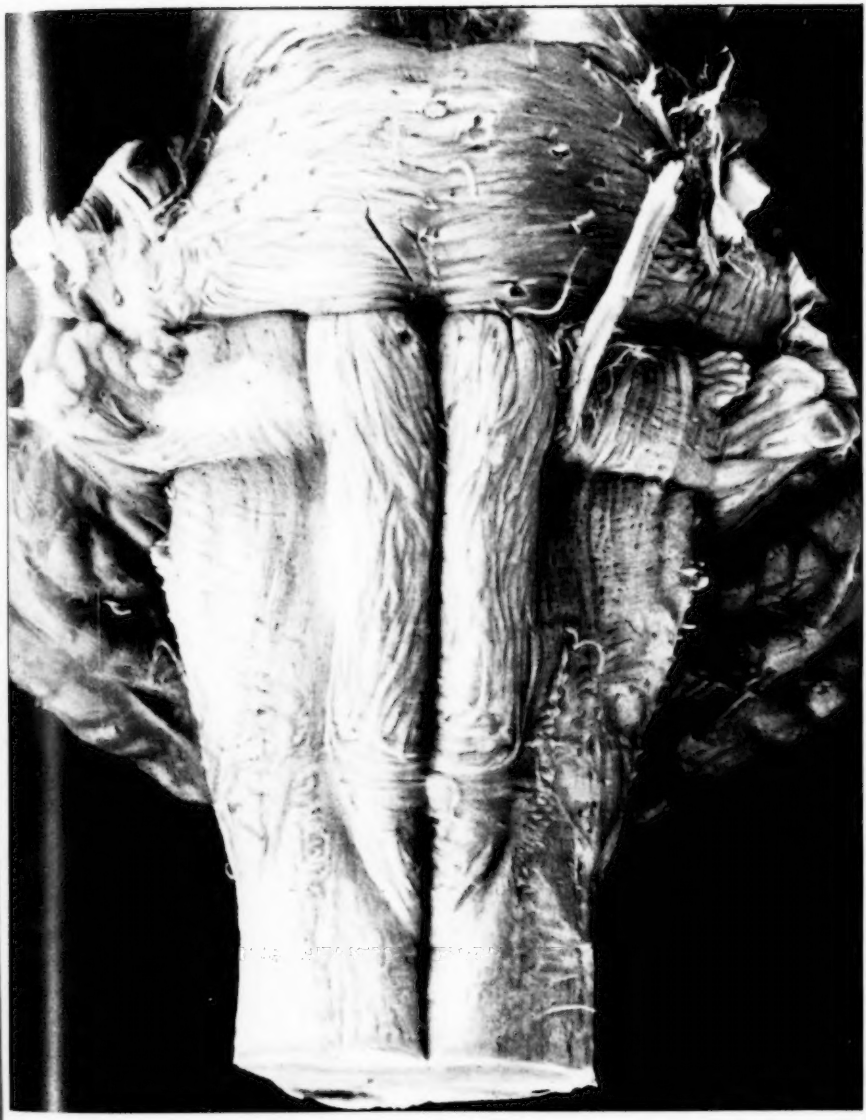


FIG. 1.—Anterior aspect of bulb, showing the ventro-lateral and arcuate fibres. Magnified 5 diam.

form body, was spread out in a thinner sheet than usual. The *ventral* cerebello-spinal tract similarly presented the appearance of more scanty and more widely separated bundles than is customarily found.

Olivary bodies.—These structures were diminished in size and the olivary fibres were poorly developed.

Cranial Nerves.

The cranial nerves showed no abnormalities in size or in origin.

Microscopic Anatomy.

Cerebrum.—The cerebral hemispheres gave no indication of morbid changes of any kind, either in the general arrangement or appearance of the cells or fibres connecting them. The optic thalamus and corpus striatum on each side were also normal.

Cerebellum.

The vermis.—In studying the cerebellum by an examination of serial sections the first point of striking import was that the vermis throughout was practically normal. In a paper of Dr. Risien Russell,[1] on the cerebellum of a puppy, to be alluded to directly, the same point appears very clearly, namely, that the mesial plane, the vermis, the phylogenetically oldest part of the cerebellum, shows the least change in these cases of defective development.

The lateral lobes.—The folia were but poorly developed. The molecular and granular layers showed no alterations in depth or in irregularity, such as were found in the puppy already referred to, and in the two cases reported by Herringham and Andrews.[2] The condition of the cells of Purkinje constituted the chief interest in the present case: they were less numerous than usual over the greater part of the lateral lobes; but more particularly was this the case in the folia on the dorsal or posterior, as opposed to the ventral or anterior, surface. In some places they were

[1] Brain, 1895.
[2] *St. Bartholomew's Hospital Reports*, vol. xxiv.

simply more diffusely scattered, in other situations they were totally absent.

These points are clearly shown in the accompanying fig. 2.

The flocculus on either side was histologically normal.

The central cerebellar nuclei appeared to be, both in general outline and in the number of cells contained, of normal structure.

Deiters' nuclei.—There was no abnormality in these nuclei, nor in the tracts connected with them.

Pontine cells.—No departures from the usual distribution were detected.

Medullary tracts. — Excepting the dorsal and ventral cerebello-spinal columns, these were of normal appearance.

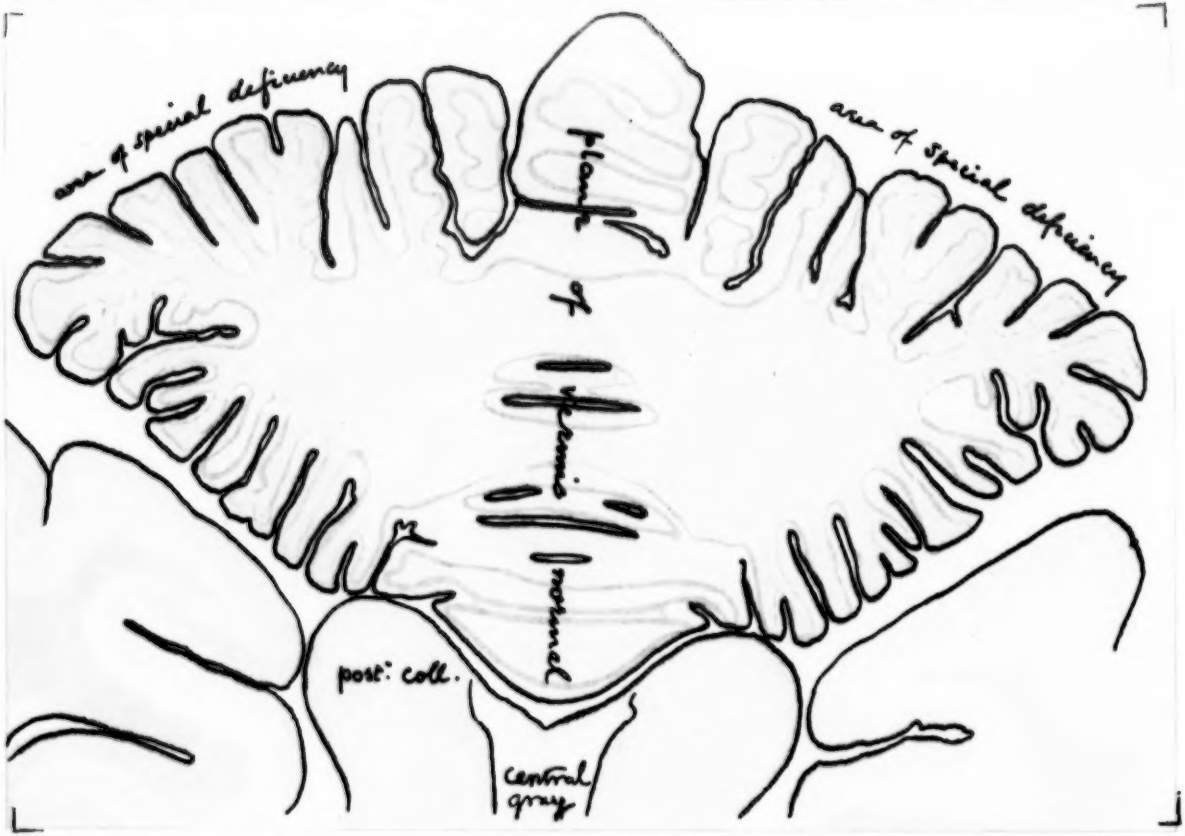
Spinal cord.—The sole abnormality discovered in the spinal cord was in the central canal, which was throughout considerably dilated, and presented the triradiate calibre of the embryonic type.

Conclusion.

It will be seen from the foregoing account that the present case clearly suggests a topographical representation of "circus movement," viz., that this abnormality of progression is associated with the lateral lobes (and so with the pontine nuclei) alone.

The case thus lends weight to the view that the cerebellum may virtually be regarded as a twofold organ, consisting of one part, the vermis, essentially spino-cerebellar, and another part, the lateral lobes, essentially ponto-cerebellar.

I take this opportunity of expressing my gratitude to Sir Victor Horsley for the generous and valuable assistance he has given me. I am also indebted to him for the figures.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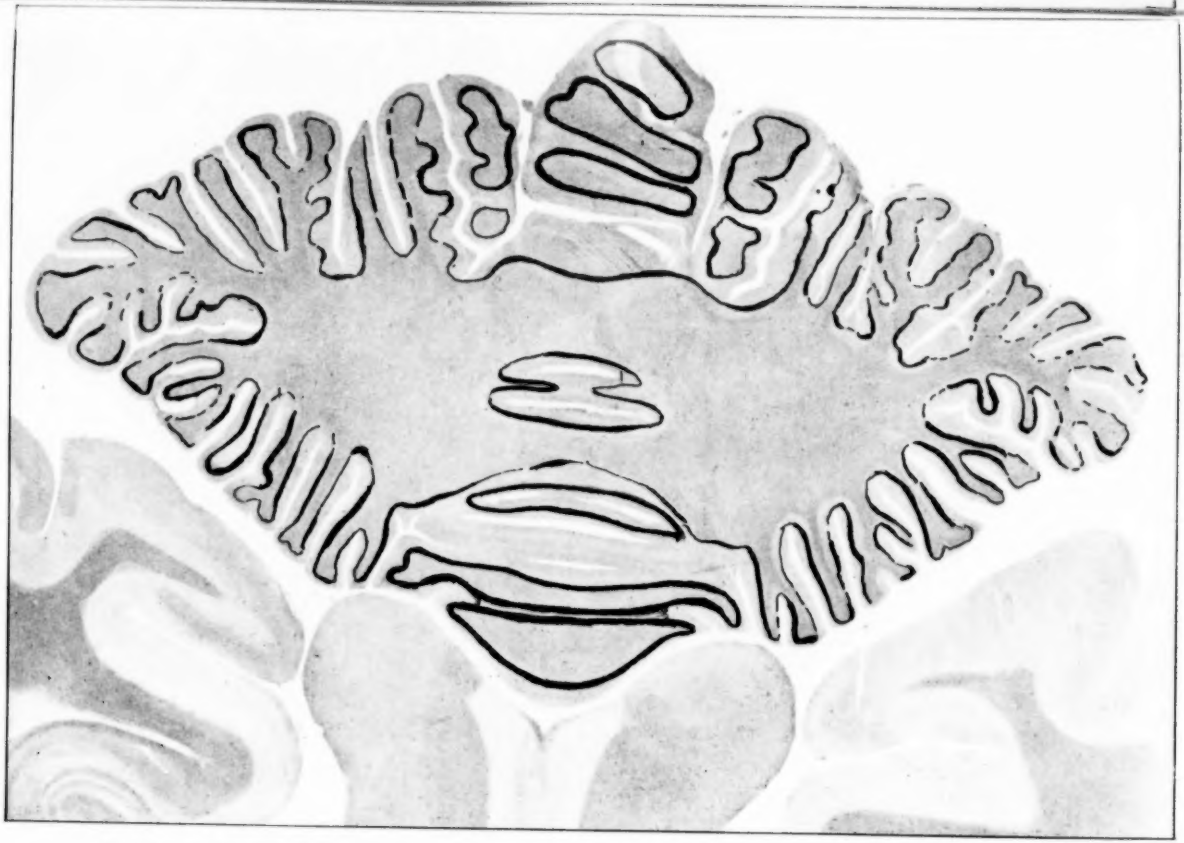

FIG. 2.—The black line indicates by its thickness the number relatively of Purkinje cells in the cortex cerebelli.

UNILATERAL CONGENITAL LESION OF MEDULLA AND SPINAL CORD; DEATH FROM PONTINE HÆMORRHAGE.

BY PURVES STEWART, M.A., M.D., M.R.C.P.

Assistant Physician to the Westminster Hospital.

A HOT-POTATO hawker, aged 41, came to the out-patient department of the Westminster Hospital on account of chronic bronchitis, the urine being moderately albuminous and with a few granular casts. It was observed that he had marked asymmetry of the muscles of the neck. He was admitted as an in-patient, and on further investigation the following history was obtained:—

No information can be had about the patient's father. He himself is the fourth of six children. Before his birth his mother, during pregnancy, is stated to have had a severe fall in the street, but at what period of the pregnancy this occurred is not known. One sister, aged 40, is deaf and dumb, one brother is alive and well, three other sisters are dead, cause unknown. The patient as a child was slow in learning to talk, nor was he able to walk alone until 4 years of age. In the lower jaw on the right side he has never had any molar teeth, either of the first or second dentition. All the other teeth erupted normally. The right side of the neck has always been noticed to be peculiar in outline.

Many years ago he had gonorrhœa, and still possesses a urethral stricture. He has been subject to chronic bronchitis for many winters.

On examination he is found to be a man of only moderate intelligence. Speech and articulation are normal, but the voice is hoarse. Smell and taste are normal on both sides. Hearing is somewhat dull in both ears, especially on the right side. The right auditory meatus is narrower than the

left, and also higher up. There is no otorrhœa. The right pinna is much smaller than the left, its lobule being particularly diminutive. The right mastoid process is absent (see fig. 1).

The optic discs are healthy, vision is acute, the pupils are equal and normal in their reactions, and the external ocular muscles are normal. The masseter and temporal muscles are strong on both sides, but, on opening the jaw, it swings obliquely to the right, owing to deformity of the mandible, which is smaller on that side (see fig. 2). All the molar teeth of the right lower jaw are absent, the canines and incisors being present. The teeth of the left lower jaw and of both upper jaws are normal in number and in development.

The facial muscles, upper and lower, are normal on both sides, the platysmæ standing out strongly on bending the head forwards.

The right arch of the palate, at rest, stands nearer to the middle line than the left, and, on phonation, the left side moves alone, pulling the raphé upwards and to the left (see fig. 4).

The right sternomastoid is absent. So also is the *portio clavicularis* of the right trapezius. This latter muscle consists only of the middle portion, taking its origin from the seventh cervical to the fourth dorsal spine, inclusive, and being inserted into the acromion process and spine of the scapula. The lower third of the trapezius, below the fourth dorsal spine, is absent (see fig. 3).

The sensibility of the mouth, pharynx, and larynx is normal. The right vocal cord is immobile in the middle line; the left cord moves freely. The right omohyoid, stylohyoid, and posterior belly of digastric are absent. The right side of the tongue is smaller than the left (see fig. 2), and the tongue on protrusion points to the right side. (Part of this deviation is due to the obliquity of the floor of the mouth from deformity of the lower jaw.) The tongue can be moved freely in all directions. The right sternohyoid, sternothyroid and thyrohyoid muscles are absent. All of the above-mentioned muscles which are

FIG. 1.

Showing mal-development of right pinna. The bifurcation of the carotid is subcutaneous. The right mastoid process is absent. The bony projection behind the angle of the jaw is the transverse process of the atlas.

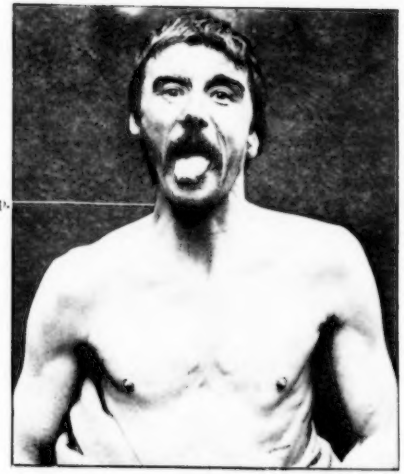

FIG. 2.

Showing deformity of right pinna and right side of lower jaw; also moderate atrophy of right side of tongue. Note absence of right sternomastoid and upper fibres of right trapezius. The right levator anguli scapulæ is subcutaneous.

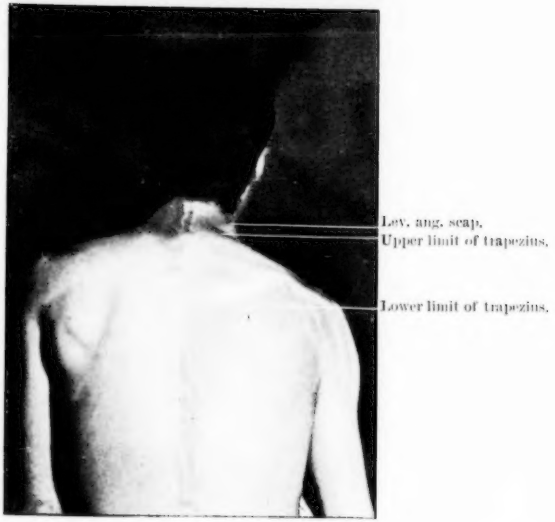

FIG. 3.

Showing downward and outward displacement of right scapula. Note parallel congenital white lines on back of neck. Right levator anguli scapulæ is subcutaneous. Right trapezius is absent above seventh cervical and below fourth dorsal spines.

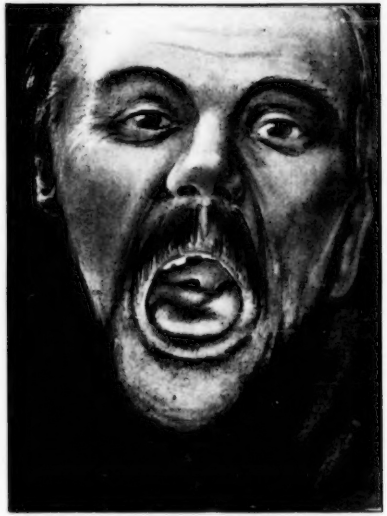

FIG. 4.

Photograph of soft palate during phonation. Note also deformity of lower jaw and of right pinna.

clinically non-active, are also non-responsive to the strongest electric stimulation, faradic or galvanic. Both sides of the tongue react normally.

In connection with the absence of the right sterno-mastoid, it is observed that the mastoid process on that side is absent and the transverse process of the atlas is subcutaneous. There is thus a deep hollow behind the angle of the right mandible, where the carotid vessels are visible, the bifurcation into internal and external carotids being easily seen (see fig. 1).

The levator anguli scapulæ is subcutaneous on the right side (see fig. 3), so also is the scalenus medius (see fig. 1). The right scapula is displaced downwards and outwards, its inner border running obliquely from below upwards and outwards, thereby rendering the outward displacement of its acromial angle still more marked. There is a very slight backward projection of the lower angle at rest.

On the back of the neck, from the level of the scalp line to that of the lowest cervical spine, there are two parallel rows of white, flattened, papillomatous projections, congenital, one row on each side of the middle line (see fig. 3).

The rhomboid muscles, the latissimus dorsi and all the other muscles of the right upper limb, together with the diaphragm, the muscles of the trunk and of the remaining limbs, are normal. The gait is normal. There is no anæsthesia. The supinator-jerks, knee-jerks, and ankle-jerks are normal, and the plantar reflexes are flexor in type. The sphincters are perfect.

In spite of the absence of so many muscles on the right side of the neck, all movements of the head and neck are executed powerfully on both sides, and there is no deviation of the head at rest.

Clinical Diagnosis.

The absence of the right sternomastoid and of part of the trapezius, together with the paralysis of the palate and vocal cord on the same side, pointed to a congenital lesion

of the spinal accessory and vagus nuclei on the right side. (This description of the spinal accessory nucleus is based on the modern nomenclature, maintaining that what was formerly termed the "bulbar part of the spinal accessory" is properly to be reckoned as part of the vagus nucleus.)

The absence of the mastoid process was probably the result of non-development of the sternomastoid muscle. The partial hemiatrophy of the tongue indicated an implication of the hypoglossal nucleus. The absence of the depressor muscles of the hyoid bone and of the posterior belly of the digastric indicated that the lesion extended down the anterior cornu of the spinal cord at least as far as the third cervical segment. This diagnosis of unilateral affection of the medulla and upper part of the cord was further supported by the maldevelopment of the right mandible and the absence of molar teeth in that bone, possibly attributable to an affection of the spinal root of the trigeminal nucleus. The other bulbar nuclei, from the facial upwards, were apparently unaffected.

The patient's unexpected death from pontine hæmorrhage, six weeks later, afforded an opportunity for pathological investigation of the case.

Subsequent history.—One morning the patient, who had not been complaining of headache or any other premonitory symptom, was sitting up in bed. Suddenly he fell back unconscious. When seen a few minutes later by the house physician, Mr. S. N. Crowther, he was found to be comatose, cyanosed, and breathing stertorously. The right arm and fingers were flaccid and motionless, whilst with the left he made semi-purposive clutching movements. The left lower limb was drawn up at the hip and knee. There was conjugate deviation of the eyes to the left. The knee-jerks were brisk and equal, there was ankle-clonus on both sides, and the plantar reflexes were extensor in type. The urine, drawn off by catheter, contained a large amount of albumen and a few granular casts. The temperature was 98° F. Two minims of croton oil were placed on the tongue.

When seen by me three hours later, the patient was still comatose and breathing stertorously, making occasional

movements with the left hand when stimulated by pinching the limb. Occasionally the right elbow made movements of extension. There was no movement of the other limbs, which were flaccid and extended at all joints. The eyes were still deviated to the left. The pupils were unequal, R. > L. semi-contracted and reacting to light. There was slight weakness of the right face, the nasolabial fold being shallower than on the left side, and the right ocular fissure wider than the left. The breathing was laboured, all the extraordinary muscles at the root of the neck being in action. The diaphragm was hardly felt to contract at all, respiration being chiefly costal in type. The knee-jerks were brisk and equal, there was ankle-clonus, R. > L., the supinator-jerks were not elicited. The plantars were now absent, also the abdominal and epigastric reflexes. The pulse was hard and bounding, 132 per minute. There was some œdema over the lumbo-sacral vertebræ, none of the ankles. The temperature was 98·4° F. Lumbar puncture withdrew clear fluid, which contained no excess of leucocytes on microscopic examination. With the sodium hypobromide test a small amount of urea was detected in the fluid.

The patient died, without regaining consciousness, twenty-four hours after the onset of coma.

Autopsy.

The heart was hypertrophied and the kidneys granular, the pelvis of the right kidney being dilated. There was some recent hypostatic pneumonia, and at the right apex some old pleuritic thickening.

All the muscles of the body were well developed, except on the right side of the neck, where there was total absence of the *sternomastoid*, *sternohyoid*, *sternothyroid*, *thyrohyoid*, *stylohyoid*, *digastric* (posterior belly), and *omohyoid*. The only muscle superficial to the carotid vessels was the platysma, which was well developed. The upper fibres of the *trapezius* were absent, the middle fibres, arising from the seventh cervical to the fourth dorsal spine inclusive, were

well developed, and below that level the muscle was absent. No signs of the spinal accessory nerve could be found. The rhomboids, levator anguli scapulæ, splenius and all other muscles of the neck were normal. The diaphragm was normal on both sides. The right *soft palate* was thinner than on the left side, and subsequent microscopic examination showed that on the paralysed side the *levator palati* was absent and the *azygos uvulæ* diminished in size. The right side of the tongue was smaller than the left. The epiglottis was bent over towards the right side and its right edge curved longitudinally.

The larynx was removed for more detailed examination. For most valuable aid in its dissection I desire to express my indebtedness to my colleague, Mr. James Black, lecturer on anatomy in the Westminster Hospital Medical School. The following are the chief points thus established:—

The thyroid cartilage, with its pomum Adami, was thrust across to the right side, giving the larynx a fictitious appearance of being more developed on the defective side. As a matter of fact the larynx was generally atrophic on the right side. This was noticeably the case with the right half of the cricoid cartilage. Subsequent dissection displayed the absence or most rudimentary condition of some of the muscles of the right side of the larynx.

Muscles.—The mylohyoid, geniohyoid and anterior belly of the digastric were about equally developed on the two sides. The posterior belly and the intermediate tendon of the last-named muscle were attenuated on the right side, and represented only by a faint strand of fibrous tissue. The right hypoglossus was well developed, but somewhat smaller than on the normal side. The right middle constrictor of the pharynx was very defective, and the styloid muscles could scarcely, if at all, be recognised. The intrinsic muscles of the tongue appeared generally fairly symmetrical on the two sides. *Muscles of the larynx.*—The right cricothyroid was totally absent. This, together with the absence of all the right infrahyoid muscles as already described, rendered the origin of the inferior constrictor of the pharynx strikingly apparent. The right

crico-arytenoideus posticus was absent, and the right crico-arytenoideus lateralis represented by a thin film of pale muscle fibres, too rudimentary to be capable of function. The small muscle described by Merkel as the kerato-cricoid, however, was present. Viewing the posterior surface of the cricoid cartilage, its asymmetry was very marked, the well-developed left crico-arytenoideus posticus presenting a noticeable contrast to the bare area of cartilage on the defective side. The arytenoideus transversus and obliquus were small but otherwise presented no feature for criticism. The right thyro-arytenoideus was but feebly developed, and this was well brought out on examining a coronal section of the larynx, when the meagre bulk of the prismatic sectional area of the right vocal cord contrasted strikingly with that of the opposite side.

Nerves.—A most careful search failed to discover any trace of the right hypoglossal nerve. The right lingual nerve was present, and a number of branches passed from it into the hypoglossus muscle (corresponding to those which usually form loops of communication with the hypoglossal nerve on this muscle). The right superior laryngeal (internal laryngeal branch), and the right recurrent laryngeal nerve, although smaller than on the left side, were recognised.

The foramen cæcum on the dorsum of the tongue was much deeper than usual. There was a well-marked lingual lobe to the thyroid gland, with a long pedicle leading upwards towards the root of the tongue, but no actual connection could be demonstrated between this pedicle and the lower end of the foramen cæcum.

The right external auditory meatus was shorter and narrower than the left, but the tympanum and auditory ossicles were normal.

The cerebrum showed no signs of disease on naked-eye examination, apart from an agonal thrombosis of the left middle cerebral artery, unassociated with any area of softening. The left cerebral hemisphere was larger than the right, weighing 18 ozs. as against 15 ozs. The left cerebellar hemisphere was also slightly wider from side to

side than the right, but not so deep from above downwards, the two halves of the cerebellum, after removal of the peduncles, weighing on the right side 2½ ozs., and on the

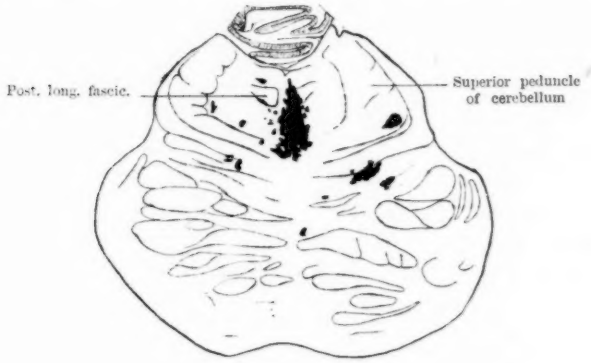

FIG. 5.

(Showing hæmorrhages in pons).

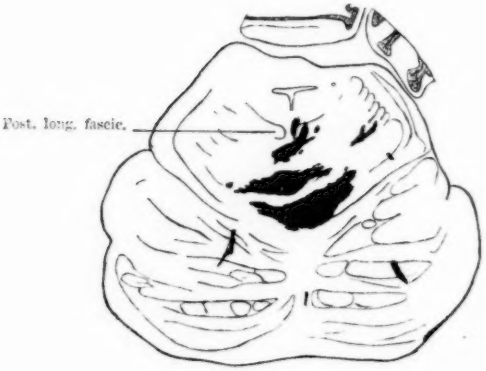

FIG. 6.

(Showing hæmorrhages in pons).

left 2½ ozs. This slight deformity of the cerebellum may have been the result of the asymmetry of the temporal and occipital bones.

The pons on naked-eye examination was symmetrical. On cutting into it, after hardening in Müller's fluid, several small hæmorrhages were found, of which the largest was about the size of a pea, in the upper third of the pons, dorsal to the transverse fibres, in the region of the fillet and posterior longitudinal fasciculus and in the intermediate area between these structures (see figs. 5 and 6). The remainder of the pons was normal and symmetrical on microscopic examination.

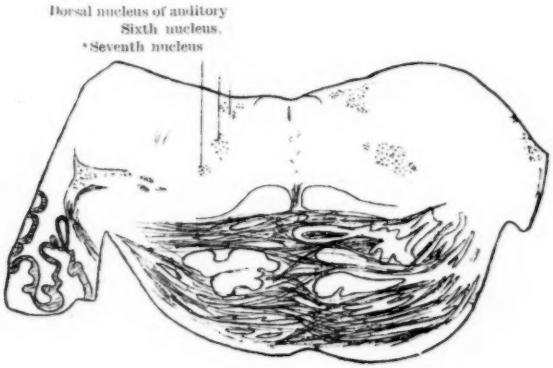

FIG. 7.

Pons.—Level of emergence of auditory nerve.

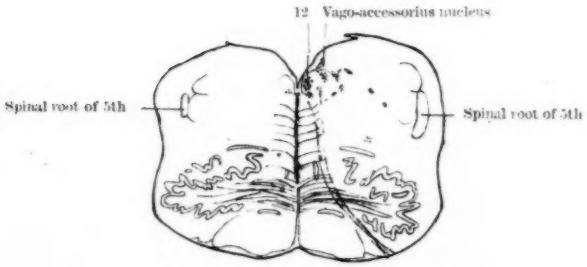

FIG. 8.

Medulla.—Upper.

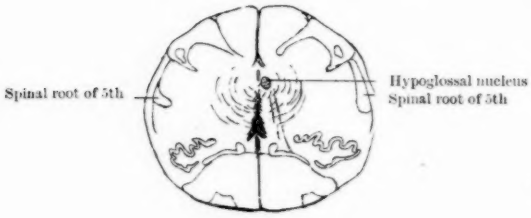

FIG. 9.

Medulla.—Lower.

The medulla oblongata showed marked abnormality, as seen in figs. 8 and 9. The olivary bodies were normal and symmetrical. On the right side the hypoglossal nucleus and the adjacent lower part of the accessorio-vagus nucleus were absent, whilst the corresponding nuclei on the left side were well developed. A few cells were present in the position of the uppermost part of the right hypoglossal nucleus, but they were of a smaller type than those of the left hypoglossal nucleus, and apparently represented part of the vagus nucleus, displaced towards the middle line. The spinal root of the trigeminus, capping the substantia gelatinosa, was much smaller on the right side than on the left. This latter abnormality did not extend down into the cord. The other nuclei of the medulla were normal, likewise the various tracts, ascending and descending.

In the spinal cord the anterior median fissure was expanded into a deep cleft, extending from the lower part of the second cervical to the upper part of the seventh cervical segment. Along the right margin of this wide fissure ran the anterior spinal artery. The cleft was lined by a thin layer of pia mater. It penetrated deeply into the gray matter on the right side, near the junction of the anterior cornu with the gray commissure (see fig. 10). The gray matter of the cord was thus much distorted, and the asymmetry could be traced upwards above the level of the actual cleft to the first cervical segment and on into the medulla, as already described. Below the seventh cervical segment the cord showed no abnormality. The

nerve-roots, anterior and posterior, were given off from the gray matter on both sides, including the cervical region. A

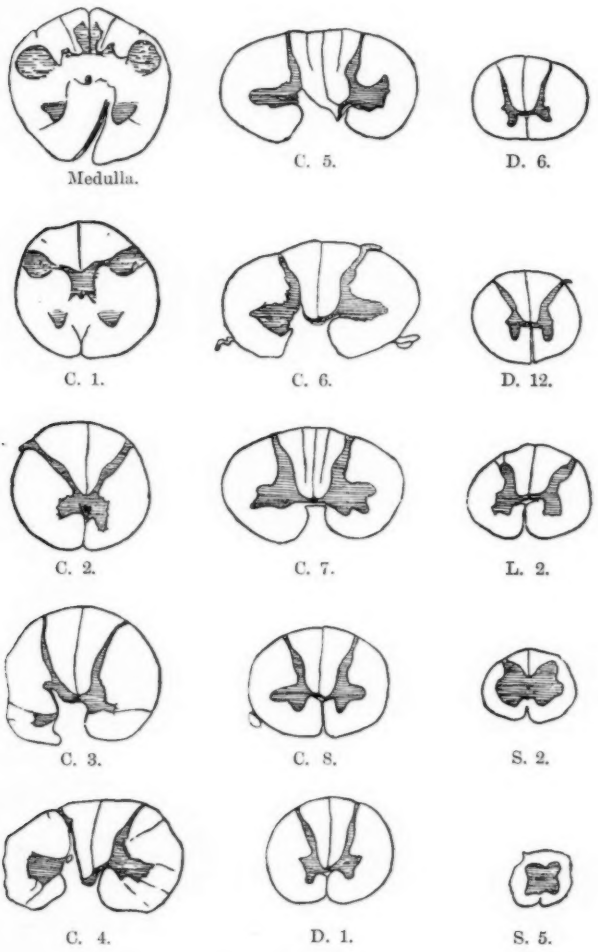

FIG. 10.

Medulla and spinal cord.

small number of nerve-cells in the right anterior cornu were missing in the position of the cleft. The distortion of the remainder of the gray matter, however, rendered it impossible to identify precisely in each segment the particular groups of cells which were absent.

The points of interest in this case, clinically and pathologically, are numerous.

Firstly, as to the fatal hæmorrhage. This, a secondary result of chronic renal disease, was situated in the tegmental region of the upper part of the pons, located almost mesially, but more extensive on the right side of the middle line (see figs. 5 and 6). It was extra-pyramidal. Clinically, the signs were somewhat confusing. The lower limbs were flaccid, yet there was well-marked ankle-clonus on both sides, together with an extensor type of plantar reflex. There were semi-purposive irregular movements of the right upper limb, and slight facial weakness on the right side, together with conjugate deviation of the eyes to the left. These two latter signs would have been explainable by a lesion implicating the right sixth nucleus. But the autopsy showed the nucleus to be intact. The ocular deviation, therefore, is more probably attributable to an interruption of the fibres connecting the right sixth with the left third nucleus, as in a somewhat similar case recorded by Raymond and Cestan (7).

The condition of the reflexes, and especially the presence of the extensor type of plantar reflex, is of especial interest, for in this case there was no interruption to the pyramidal tracts in any part of their course, the hæmorrhage being situated posteriorly in the tegmentum. Kornilow (5) has recently maintained that the Babinski phenomenon can occur independently of a lesion of the pyramidal tracts, and this case supports such a view. The explanation of its occurrence, however, remains obscure. Another unusual feature was the absence of the high temperature which so frequently occurs in fatal pontine hæmorrhages. Nor were the pupils contracted.

Even more remarkable is the unilateral lesion in the

medulla oblongata and upper part of the spinal cord. The condition was congenital and non-progressive. It does not correspond to any variety of syringomyelia. I have been unable to find in the literature of nuclear lesions of the medulla or cord the record of anything precisely similar. The two parallel white lines visible in the skin of the back of the neck (see fig. 3), were probably associated developmentally with the lesion of the brain-stem. They may be regarded as analogous to the mole in the lumbar region, so commonly seen in cases of spina bifida occulta.

Only a small number of cases (chiefly of syringomyelia) have been published, *e.g.*, those of Schmidt (10), Kretz (6) and Weintraud (14), in which the diagnosis of nuclear paralysis of the accessorius was made, and in none of them was the case completed by subsequent pathological investigation.

In our case the lesion of the central nervous system was an extensive one, implicating the gray matter of the following motor nuclei :—vagus, accessorius, hypoglossal, and anterior cornu as low as the sixth cervical segment. Together with these, the spinal root of the trigeminus was also affected. The other cranial-nerve nuclei, from the facial upwards, were unaffected. On the affected side the spinal accessory and hypoglossal nerves were absent, whilst the anterior cornu, though distorted in shape and diminished in size, gave off its anterior nerve-roots throughout the affected region. The vagus trunk was present, though smaller on the affected side, its superior laryngeal and recurrent laryngeal branches being especially reduced in size.

This case is also of value in regard to the innervation of of the trapezius and as to the parts played by the spinal accessory and by the cervical nerves respectively. In our patient the spinal accessory nerve was absent, the cervical nerves present. Associated with this there was total absence of the sternomastoid and of the upper and lower thirds of the trapezius, whereas the middle third of the trapezius was well developed. This indicates a separate innervation of the middle third of the trapezius by the cervical anterior

roots, the remainder of the muscle belonging to the territory of the spinal accessory. Such a view harmonises with the opinion of Remak (8). Gowers (4), in his latest edition, states that the whole of the trapezius is supplied by the spinal accessory, and that the twigs from the cervical plexus merely pierce the muscle without supplying it. Schulz (11), again, from a study of a large number of recorded cases, comes to the conclusion that the result of a pure lesion of the spinal accessory, without implication of the cervical plexus, is complete palsy of the lowest third and partial palsy of the upper two-thirds, the clavicular fibres being more severely paralysed than the acromial. The apparent divergence of these views is probably to be explained by variations in the proportional territory of the spinal accessory in different cases. Thus, for example, Schlodtmann (9) records an interesting case of unilateral paralysis of the palate, larynx, sternomastoid and trapezius, the result of a fracture of the base of the skull, without implication of the cervical roots, and in which there survived only a few strands of the acromial bundle, whose total breadth was about that of a pencil.

The characteristic deformity occurring in most cases of trapezius paralysis was clearly described by Duchenne (3), who applied to the displacement of the scapula the term "*mouvement de bascule*," and stated that the occasional absence of this deformity in certain cases was explainable by survival of the acromial bundle, quite a small strand of this bundle being sufficient to prevent the displacement. But in our case the acromial bundle was well developed, and yet the scapular displacement was perfectly evident.

As to the sternomastoid, it should be noted that in our case its absence produced neither deformity at rest nor impairment of the movements of the head and neck. This exemplifies the truth of Gowers' dictum (4) that there is no such thing as a "paralytic" torticollis, though Bernhardt (2) in his recent text-book states that unilateral paralysis of this muscle causes moderate deviation of the head towards the affected side, the chin being slightly raised. In our case no such deviation occurred.

Similarly, the absence of all the infrahyoid muscles produced no impairment of movement of the hyoid bone, though the larynx was distorted towards the affected side.

It is interesting to note that, in spite of the absence of the right hypoglossal nucleus and nerve, the corresponding hemiatrophy of the tongue was only moderate in degree. The intrinsic muscles of the tongue were certainly smaller on the right side than on the left, but the tongue itself was not curved longitudinally as in ordinary hypoglossal palsy, nor were its electrical reactions different on the two sides. Moreover the orbicularis oris was unaffected, contrary to what is usually found in bilateral lesions of the hypoglossal nuclei.

The explanation of these facts is somewhat difficult. Most likely the hypoglossal nucleus of the healthy side controlled both halves of the tongue. (The right and left nerves, according to Thane (13), are occasionally connected by a cross branch or loop in the substance of the genio-hyoid.) Another less probable view might be that, inasmuch as the lingual nerve supplied branches which entered the hyoglossus, the motor fibres of the trigeminus may have taken on a vicarious distribution.

Nor is the question appreciably enlightened by a consideration of the absence of the depressor muscles of the hyoid bone. Whichever nerve assumed the vicarious function of innervating the right half of the tongue, did not reach the infrahyoid muscles. These latter are commonly believed to be supplied, not by the hypoglossal itself, but by branches from the first and second nerves forming the loop of the *ansa hypoglossi*. Thus Beevor and Horsley (1), who stimulated the intracranial roots of the hypoglossal in the monkey, obtained no contraction in the infrahyoid muscles, which responded briskly, however, to stimulation of the first and second cervical nerves. Yet Stierling (12), on the other hand, records a case of annular fracture of the anterior part of the foramen magnum in man, implicating the hypoglossal nerve close to its origin, in which, together with paralysis of the vagus on that side, there was paralysis of the corresponding half of the tongue and of the depressors of the hyoid bone on the same side, the cervical roots being stated to be uninjured.

In our case there was a deep fissure in the anterior cornu, which was most probably causally connected with the absence of the infrahyoid muscles. And yet all the anterior roots were present in the cervical region, and, moreover, were efficient in supplying the other deep muscles of the neck, together with the middle third of the trapezius. Of course, it must be remembered that the nucleus of the spinal part of the trapezius lies lower in the cord than that of the infrahyoid muscles.

The atrophy of muscles in the larynx is undoubtedly to be associated with the lesion of the vagus, the lower part of whose nucleus was deficient, and whose trunk was much diminished in size, especially its superior laryngeal and recurrent laryngeal branches. Of the laryngeal muscles certain were absent (crico-thyroideus and crico-arytenoideus posticus); others were so rudimentary as to be incapable of function (crico-arytenoideus lateralis); others, again, were simply diminished in size (arytenoideus and thyro-arytenoideus). The position of the vocal cord corresponded to that usually seen in total recurrent laryngeal palsy, but there could be no question of contracture of abductor or adductor muscles, since the crico-arytenoideus posticus was absent and the crico-arytenoideus lateralis reduced to a rudimentary strand. The position of the cord was simply that of the edge of the thyro-arytenoideus muscle, which supported the cord immovably in a semi-abducted posture.

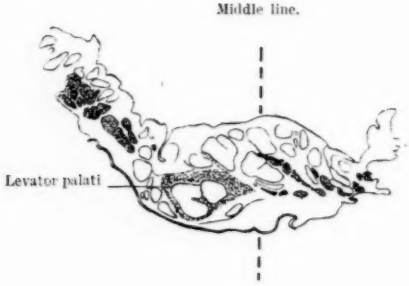

FIG. 11.

Soft Palate.—Showing absence of levator palati on right side.

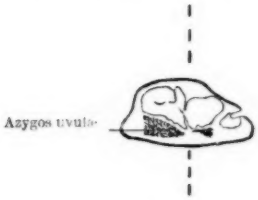

FIG. 12.

Uvula.—Showing atrophy of azygos uvulæ on right side.

In the soft palate the right levator palati was absent, and the right azygos uvulæ was much smaller than the left (see figs. 11 and 12), whilst in the pharynx the right middle constrictor was greatly diminished in size. This corresponds with remarkable accuracy to a lesion of that part of the vagus formerly named the "bulbar part of the spinal accessory." Beevor and Horsley (1) have shown experimentally in the monkey that the motor fibres for the levator palati and azygos uvulæ, as well as part of the constrictor muscles of the pharynx, are thus supplied.

The deformity of the temporal bone and lower jaw is also deserving of notice. Absence of the sternomastoid is sufficient to account for the rudimentary condition of the mastoid process. The smallness of the lower jaw, however, cannot be explained as the result of muscular atrophy, since all the muscles attached to the mandible were present and well developed. Probably the condition of the lower jaw is to be associated with the congenital smallness of the spinal root of the trigeminus. Such a view is corroborated by the absence of the lower molar teeth and by the smallness of the external auditory meatus and pinna on the affected side. The external auditory meatus, pinna, middle ear and Eustachian tube are all developed from the first visceral cleft, whilst from the mandibular arch, between this cleft and the mouth, the lower jaw is developed. And all these structures lie within the territory of the trigeminal nerve.

To Dr. Ferrier I desire to express my indebtedness for

his permission to conduct the microscopical portion of the above research in the neuro-pathological laboratory of King's College.

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BINOCULAR AND STEREOSCOPIC VISION IN MAN AND OTHER VERTEBRATES, WITH ITS RELATION TO THE DECUSSATION OF THE OPTIC NERVES, THE OCULAR MOVEMENTS, AND THE PUPIL LIGHT REFLEX.

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THE work on which this paper is based has been done at intervals during the last seven years, and consists partly of the results of a series of experimental lesions of the optic tract on one side in cats; of division of the posterior commissure in cats; and the division of one optic nerve in pigeons, owls, rabbits, and cats, followed by microscopical examination by the Marchi method of the optic chiasma and optic lobes in these animals, and by the Weigert-Pal method in the toad and chameleon. In addition, I have studied the pupil reaction to light, the position of the eyes in the head, and the ocular movements in a considerable number of animals, and the conclusions at which I have arrived I have drawn up in the following summary, as a preface to the paper.

SUMMARY OF CONCLUSIONS.

(1) The decussation of the optic nerves at the chiasma is complete in all fishes, amphibia, reptiles and birds, whether possessed of binocular vision or not.

(2) Binocular vision is originally associated with carnivorous habits, and is found to a moderate degree amongst carnivorous fishes in a few of the sharks and rays, in some amphibia, as the toad, which lives on flies and insects, and

in many carnivorous birds, especially the larger gulls, some penguins, hawks, owls and vultures. Amongst mammals binocular vision is especially developed in the carnivora and in the primates. In the latter group of monkeys and man perfect binocular vision is probably associated with the development of the hand as a prehensile organ.

(3) Though many of these animals have fair binocular vision, yet in all vertebrates below mammals there is total decussation of the optic nerves at the chiasma, and their vision is therefore not stereoscopic in the same sense as in the higher mammals, in which visual impressions from the two eyes are received on the same side of the brain, owing to the semi-decussation of the optic nerves at the chiasma; and their macular vision is also less developed than in the higher mammals, as in the felidæ and primates.

(4) In animals with laterally-placed eyes and periscopic vision the movements of the eyes are independent of each other, as seen typically in the chameleon, while conjugate movements of the eyes for lateral and vertical movements are associated with the acquirement of stereoscopic vision. Convergence of the eyes in the act of feeding is seen in many animals with total decussation of the optic nerves and otherwise independent movements of the eyes, as in the chameleon and the hornbill.

(5) The reflex contraction of the pupil to light is limited to the eye stimulated, and is not consensual, in amphibia and in birds, whether possessed of binocular vision or not. In the rabbit, with scarcely any binocular vision, only the one pupil reacts, and the decussation is almost complete. In cats and in the higher mammals with good binocular vision and semi-decussation of the optic nerves at the chiasma there is consensual pupil reaction. Consensual pupil contraction to light is therefore dependent, not on binocular vision alone, but on semi-decussation of the nerves at the chiasma.

(6) In the rabbit and the cat both the crossed and the uncrossed fibres are spread equally through all parts of the two tracts, and are not collected into any distinct bundle.

(7) Experimental division of the optic tract on one side

in cats produced distinct homonymous hemianopia, with the hemianopic pupil reaction, and with considerable diminution of the pupil reaction to direct light in the opposite eye, though its consensual reaction was brisk on exposure of the other eye to light.

(8) Injuries, either lateral or dorsal, of the anterior part of the anterior corpus quadrigeminum in cats, if deep enough to wound the tegmental roof surrounding the central gray matter, have produced in four cases descending degeneration in Meynert's fountain decussation, most of the fibres decussating at the level of the third nerves to form a longitudinal tract immediately ventral to the opposite posterior longitudinal bundle, but a few uncrossed fibres passing into the posterior longitudinal bundle of the same side. These are the antero-lateral columnar fibres already traced by Boyce into the lower cervical cord. In no case have I produced any degeneration in Forel's lower decussation.

(9) In animals with divergent optic axes and periscopic vision and total decussation of the optic nerves there must exist a posterior total decussation of the pupil reflex fibres connecting the anterior corpora quadrigemina with the third nuclei; while in mammals with good binocular vision and semi-decussation of the optic nerves at the chiasma there must be a semi-decussation of the posterior pupil reflex fibres. Consensual reflex contraction of the pupils to light is effected by means of this posterior semi-decussation between the corpora quadrigemina and the third nuclei, and not by means of any commissural fibres between the sphincter pupillæ centres in the third nuclei.

(10) The lesion in typical Argyll-Robertson pupil is probably a sclerosis of these fibres, some of the fibres of Meynert's decussation, and simple reflex iridoplegia is not due to a nuclear lesion, though a nuclear lesion may be superadded in the dilated type of Argyll-Robertson pupil, in which the reaction to convergence and accommodation is also lost.

I have divided my paper under six headings:—

(1) Binocular vision, and its relation to the decussation of the optic nerves, and to the feeding habits of the animal.

(2) Stereoscopic vision.

(3) Macular vision.

(4) Ocular movements, conjugate and independent, and their relation to stereoscopic and periscopic vision respectively.

(5) The path of the pupil reflex to light, including the results of experimental section of one optic nerve, one optic tract, and of the posterior commissure in cats.

(6) Argyll-Robertson pupil.

The experimental part of this work was commenced in 1897, in order, by studying the path of the pupil reflex to light in various animals, to afford some help in elucidating the problem of reflex iridoplegia, or the Argyll-Robertson pupil. Incidentally, some other interesting facts have been observed, bearing on the relation of binocular vision in birds and animals to their feeding habits and mode of life. Some of the contents of this paper were read at a special meeting of the Neurological Society on December 21, 1899, when the microscopical sections relating to the experimental part of the work were shown. Publication has been delayed until now in the hope of rendering the work more complete in a larger series of animals; especially I wished to obtain a specimen of the optic chiasma of the fox shark, *Alopecias vulpes*, sometimes known as the thresher, which is remarkable amongst elasmobranchs for possessing a fair degree of binocular vision, the eyes being set far forwards on the head. I have not, however, been able to obtain a specimen. An ideal experiment would be an experimental enucleation of one eye in such an animal, with subsequent tracing of the degenerated nerve-fibres through the optic chiasma and tracts, by Marchi's method, as Krause (14) did with goldfish. The difficulties of attaining this end, however, appear to be even more insuperable.

Binocular Vision.

Knowing the rashness of assuming anything to be true without proof, yet, in the absence of direct evidence, I think it may be safely assumed that the decussation of the optic

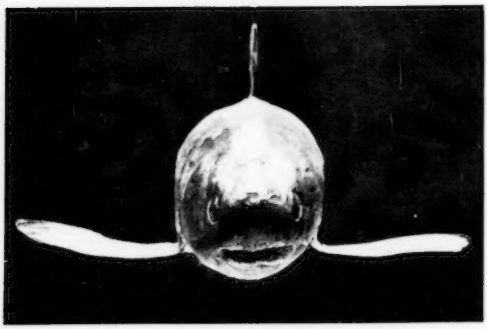

FIG. 1.

Front view of fox shark, *Alopecias vulpes*.

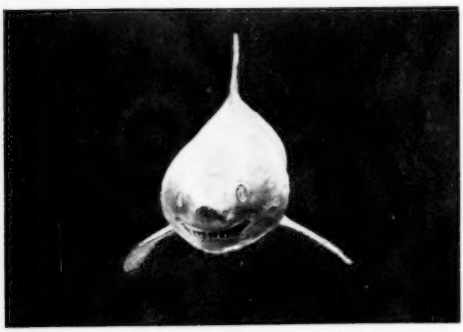

FIG. 2.

Front view of Porbeagle shark, *Lamna cornubica*.

nerves in elasmobranchs with binocular vision is as complete as in those with laterally placed eyes and periscopic vision, as the dog-fish and skate, just as in those birds which possess binocular vision, the decussation of the optic nerves is as complete as in those with laterally placed eyes. I would here remark that binocular vision does not necessarily indicate the presence of semi-decussation of the optic nerves at the chiasma, as is stated in "Foster's Physiology." Foster says (8), "The amount and character of the decussation taking place in the optic chiasma differs in different animal types, the difference having relation to the amount of binocular vision, which in turn depends on the position of the eyes in the head; that is, on the prominence of the face between the eyes. In the fish, for instance, with laterally placed eyes, no binocular vision at all is possible, and the decussation is complete; the whole optic nerve of each eye crosses over to the other optic tract."

Now this statement, though in the main true for mammals, is not true for many birds and some fishes. In the lowest fishes, the cyclostoma, which include *Petromyzon* and *Myxine*, the lamprey and the hag-fish, the optic nerves do not cross the middle line, but each arises from the corresponding side of the fore-brain, the nerves passing to the eyes without decussation. It is true that in all the Teleostean fishes the eyes are either set laterally or back to back, no attempt at binocular vision being possible, but in some of the elasmobranchs, as the fox shark and the Porbeagle shark, *Lamna cornubica*, the eyes are set well forward, and these animals must possess a considerable degree of binocular vision (see figs. 1 and 2). These photographs I took, with the permission of the Secretary, from stuffed specimens in the Fish Gallery of the Natural History Museum, and they have not therefore the value as evidence of fresh specimens, but all four of the specimens of the thresher, or fox shark, in the Gallery, show the same point in greater or less degree. The specimen of the Porbeagle shark is recently stuffed, and Dr. Ridewood, of the Natural History Museum, who examined the specimen with me, considers it a very fair example of the true shape of the

animal. Stuffed specimens are apt to be deceptive in regard to the true position of the eyes of the animal during life, a greater or less amount of stuffing of the head altering the angle of apparent vision considerably. Making all due allowance for this, however, it seems clear that partial binocular vision must be admitted for these species of elasmobranch fishes.

The presence of binocular vision in animals seems to me to depend upon their feeding habits rather than upon their social position, if I may use the term, in the animal scale. Carnivorous animals, which have to follow up and catch their prey, which is likely to be dodging hither and thither in the attempt to elude their captor, require especially keen sight for this purpose, and it is manifestly an advantage for them to be able to keep both eyes fixed at the same time upon the moving object. To effect this, their eyes are set well forwards, allowing of more or less binocular vision. Amongst fishes this arrangement is found in only a few of the elasmobranchs, carnivorous fishes with rapid power of locomotion. It is not found by any means in all of these carnivorous fishes, even among the fiercest and most active, such as the blue shark, *Charcarodon*, and in the more slowly moving skates and rays the eyes are set completely back to back. It seems not improbable that in this primitive group of vertebrates the extra value of binocular vision to a carnivorous animal, which has to pursue and catch its prey, is only commencing to be recognised, a few types only having acquired it, and that to an imperfect degree. Amongst the amphibia a higher degree of binocular vision is seen in the frogs and toads, the total divergence of the optic axes varying from about 135° in *Rana clavata* to 120° in the bull frog, while in the giant toad, *Bufo marinus*, the angle is as small as 90°.

In the chameleon is seen an extraordinary combination of completely independent movements of the two eyes for all ordinary purposes of vision, such as when seeking for its prey (flies, grubs, worms, &c.), with the most perfect power of convergence of the eyes, with binocular vision, in the act of striking its prey. This animal has the most

extended range of movement of the eyes of any animal I have studied. Each eyeball is almost completely enclosed in a casing of the ordinary horny epidermis which covers the whole animal, leaving only a small round aperture in front, through which can be seen, deeply placed, the small pupil. The visual field of each eye must be therefore very small and constricted, but to make up for this the range of movement of the eyes is very great, the eyeball being capable of being turned through about 180° in the horizontal plane from directly forwards to a backward direction, and through about 90° in the vertical plane. The eyes move in a slow, jerky fashion, recalling the movement of a searchlight, while the visual field of each eye must resemble the limited field illuminated by a searchlight, on account of the deep setting of the eye. Except when the animal is in the act of striking its prey, when fairly active, the eyes are continually being moved about, but quite independently, one eye perhaps looking forward, and the other pointed downwards and backwards. Under these circumstances I have never seen either eye point further forward than up to 15° of the antero-posterior line, and thus I first came to the erroneous conclusion that this animal had no power of binocular vision, since the animal I first studied would not feed, it being then late in the autumn. I have, however, since studied the movements of the eyes in a feeding animal, and it was a great surprise to me to see how perfect a power of convergence of the eyes the chameleon possesses. If, while the animal is at rest, and the eyes are roving in different directions independently as usual, a mealworm is offered within striking distance, that is to say, about the animal's own length away, presently one eye will catch sight of it, and if the bait is acceptable the head will be slowly turned towards it, and both eyes will be brought to bear upon the morsel, actually converging directly upon it. The mouth is seen then slowly to open slightly, and the moist tongue shows between the animal's jaws, when, like a flash, the tongue is thrown out and seizes the worm, and is retracted again into the mouth. The first part of the operation is performed slowly, with the deliberation charac-

teristic of the movements of the animal, while the last act of the drama is startling in its rapidity. In the accompanying photographs (see figs. 3 and 4), taken from an animal in the reptile house of the Zoological Gardens, through the kindness of the superintendent, Mr. R. I. Pocock, two views of the chameleon are given, fig. 3 before food is presented, showing the eyes looking in opposite directions, and fig. 4 just before the act of striking with the tongue, showing the eyes focussed in convergence upon the object. Such a combination of independent movement of the eyes with perfect power of convergence and binocular vision is most remarkable, and is probably best explained by a reference to the animal's peculiar habits. All its movements of locomotion are slow and very deliberate, and the extensive range of movement of the eyes is necessary to make up for the small field of vision of each eye owing to its encasing in the horny integument. In the independent movement of the two eyes, exploring the area on each side of the animal in its search for food, the same purpose is served as in an animal such as the crocodile, in which the eyes are placed back to back, with much less power of movement, but with much larger ocular apertures in proportion, and therefore a corresponding larger visual field for each eye. The crocodile, however, has no power of binocular vision, though it is carnivorous in its habits, for being slow and clumsy in his movements, and unable to pursue and catch rapidly moving animals, it is probably of greater advantage to him to have more range of vision, as is obtained with laterally placed eyes, than the accuracy of sight accompanying binocular vision. Being an amphibious reptile, his vision cannot be equally acute in air and in water, owing to the curvature of the cornea, an additional reason for the less accurate periscopic vision. His method of taking his prey, too, is different essentially from that of the chameleon. The latter animal, having sighted his prey, has to get within range so that he may shoot his tongue out to catch it, a distance perhaps equal to his own length. He has therefore to get his range accurately, that he may make certain of taking his prey at the first attempt, since a second oppor-

FIG. 3.

Chameleon, back view, showing the right eye turned directly backwards towards the camera.

FIG. 4.

Chameleon, front view, showing the eyes converged on a mealworm (not shown in photo), while in the act of striking with the tongue.

tunity is not likely to be afforded him under natural conditions, and it is obvious that the focussing of both the eyes upon the object, giving him binocular vision, is of great advantage to him in the accomplishment of this accuracy of range finding.

Although this degree of binocular vision is found amongst certain of the amphibia and reptiles, yet the optic nerves in these animals decussate totally at the chiasma, as I have shown by cutting sections of the chiasma in a toad and in chameleon (fig. 5). In birds, much greater variations are met with in the positions of the eyes in the head than is seen in reptiles, amphibia and fishes. In graminivorous and fruit-eating birds, as the parrots, pigeons, fowls,

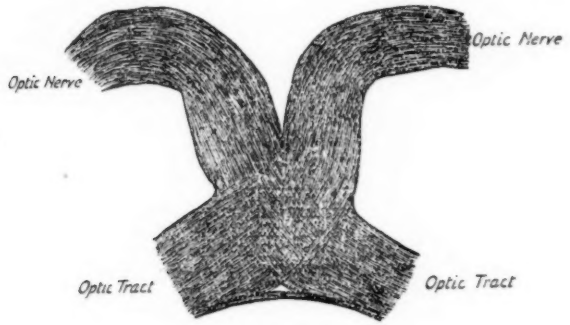

FIG. 5.
Section of chiasma of chameleon, stained by Weigert-Pal method.

ducks, swans, many finches, and others, the eyes are set laterally on the head, no attempt at binocular vision being possible. Indeed, when such a bird attempts to look at any object carefully, it always holds its head, as it were, on one side. Birds more omnivorous in their feeding, such as the robin, thrush, and sparrow, have slight binocular vision, the eyes being set more forward. It is in the birds of more purely carnivorous habits, which require especially keen sight to follow up their prey, that the highest degree of binocular vision is seen, with the eyes set further forward, and more side by side. Amongst fish-eating birds this is

well seen in the heron, kingfisher, bittern, puffin, penguins, and the larger gulls. Indeed, the gannet, of the fish-eating gulls, and the thick-billed penguin have as much binocular vision as some owls. Considerable binocular vision is also seen in the shrike, or butcher-bird, and in the goat-sucker, or night-jar. This latter bird has a head very like a small owl, and it also resembles the owl in its feeding habits, preying on moths and cockchafers in the dusk of twilight. The class of birds which shows the most consistent amount of binocular vision is the raptatores, or the hawks and harriers, vultures and owls, and of these the owls have by far the greatest degree of binocular vision; in some of them, as in the great eagle owl, *Bubo maximus*, the total divergence of the optic axes is no greater than 45°. The reason for this great development of binocular vision in the owl is apparent when we compare his feeding habits with that of other raptatores. Watching in the dusk for field-mice and other small animals on which he preys, the accuracy of sight and range due to his great binocular vision must be of immense advantage in enabling him to follow the quick, jerky movements of such small mammals, and to pounce upon his prey with unerring accuracy in the dim light of twilight. Although the vision of a pigeon and of an owl is so different in character, the one having laterally placed eyes, an extended range of vision on both sides, and no binocular vision, while the latter has the eyes placed right forward, with accurate binocular vision, but with a less extended range, yet the decussation of the optic nerves is complete in both cases. In the pigeon I have followed the path of the degenerated optic fibres through the chiasma after enucleating one eye, and a fortnight later killing the bird, and cutting sections of the chiasma after staining with Marchi's method, which showed that all the degenerated fibres crossed over into the optic tract on the other side (fig. 6). I attempted the same experiment in the owl, but failed three times, the bird in each case not surviving the operation long enough for the characteristic degeneration to take place. I therefore stained sections of the chiasma by Pal's method, which show quite clearly that the decussa-

tion of the fibres must be complete; the angle of entrance of the optic fibres being too sharp to suggest the possibility of any of them turning back into the tract of the same side, and all the visible bundles, though interlacing with those of the other optic nerve, appear to pass across into the opposite optic tract.

Turning to mammals, we find here again a similar variation in the position of the eyes in the head, laterally placed eyes being found for the most part in the herbivora and rodents, that is to say, in animals whose food does not run away from them, whilst the most perfect binocular vision is found in the carnivora, especially in the cats, and in the

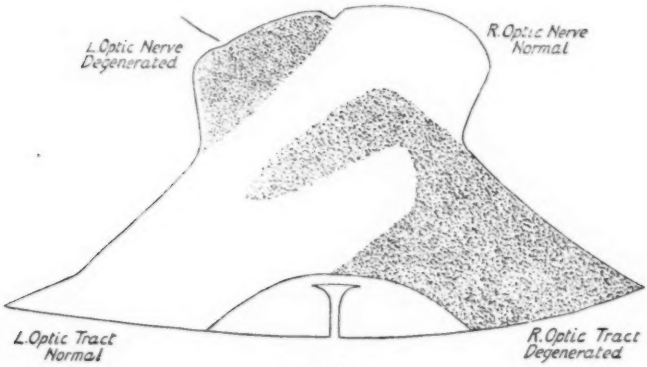

FIG. 6.

Chiasma of pigeon, stained by Marchi's metheod, after enucleation of the left eye.

primates. Taking the rabbit or hare as a type of a rodent, its eyes are set quite laterally, with practically no binocular vision possible, giving the animal a wide range of vision on either side, which, as Lindsay Johnson suggests (12), must be of great service in giving early warning of the near approach of possible enemies. Yet this great range of lateral vision makes of necessity vision in the direct line ahead correspondingly bad, and it is well known how a rabbit or hare may run straight towards a gun, through inability to perceive the danger that lies directly in front of him. Associated with this lateral or periscopic vision is a

practically complete decussation of the optic nerves at the chiasma. This I have examined both in the ordinary way by sections stained with hæmatoxylin by Pal's method, and also after experimentally enucleating one eye and a fortnight later staining with Marchi's method to trace the degenerated fibres. The Pal sections appeared to show total decussation, none of the fibres being seen to turn into the optic tract on the same side, though the angle formed by the optic nerves

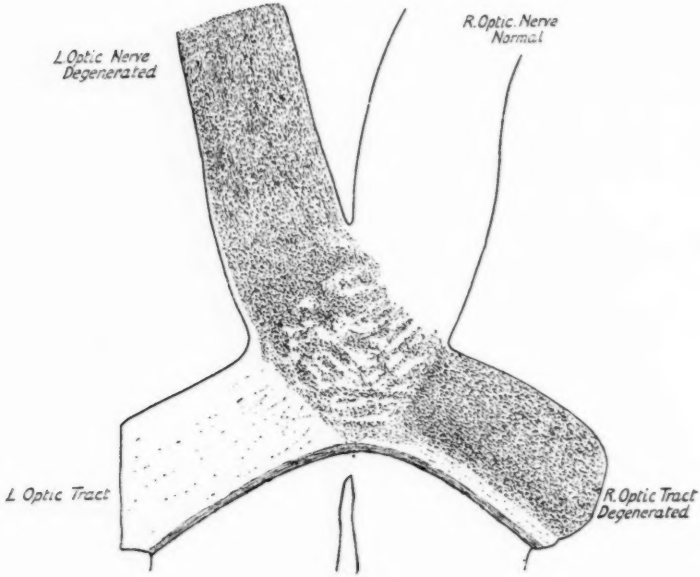

FIG. 7.

Section of chiasma of rabbit, stained by Marchi's method.

and the tracts at the chiasma is very much wider than in the owl, so that it is easy to conceive that in the rabbit a few fibres may turn into the optic tract on the same side. This indeed the Marchi sections show (fig. 7), as has also been shown by Ramón y Cajal (4), whose figures of the rabbit's and cat's chiasma stained by Marchi's method are exactly like the sections I exhibited at the Neurological

Society in 1899. According to Singer and Munzer (16) the decussation is complete in mice and rats.

Amongst the ungulata a certain amount of binocular vision is the rule, as in horses, oxen, antelopes and deer. The divergence of their optic axes is, however, great, and their acuity of vision is probably not very good, their senses of smell and hearing being proportionately keener. In these animals, although the binocular vision is not so well marked as in most of the owls, yet the movements of the eyes are mainly conjugate, not independent as in birds, and the decussation of the optic nerves is not complete, a small bundle turning into the optic tract of the same side. This has been shown in the horse and calf by two Vienna observers, using Marchi's method. Practically perfect binocular vision is seen amongst the feline group of the carnivora, cats, lions, leopards and tigers, predatory animals whose method, like that of the owl, is to lie in wait and pounce on their prey in the dim light. Other members of the carnivora, such as the dogs and foxes, bears and otters, on the one hand, and the seals, walrus and sea-lions on the other, have rather less marked binocular vision than the cats, the face protruding more between the eyes; this is, perhaps, most marked in some of the frugivorous bears. Anyone who has observed the habits of cats and dogs will, I think, admit that the cat has far the keener vision of the two, the dog depending more on scent. An additional point which confirms me in this opinion is the sluggishness of the pupil reaction to light in the dog as compared with the brisk contraction of the cat's pupil on exposure to light. This sluggishness of the pupil reaction to light is also seen in herbivora and rodents, such as horse, ox, rabbit, and it is almost certain that in these animals keenness of sight is less well developed than their acuity of hearing and smell perception.

The highest development of binocular vision is found in the anthropoidea, man, apes, and monkeys. In the lemurs it is less well developed, the face protruding more between the eyes, like that of a dog. It is only in the group of anthropoidea that high development of binocular vision is found

unassociated with specially carnivorous habits, and its acquirement in such high degree is probably associated with the development of the hand as a prehensile organ, objects seized being submitted to the test of inspection rather than that of smell, a special sense that is becoming atrophic in the primates. Binocular vision is clearly of great assistance in the accurate use of the hand for fine movements, which, in proportion to its specialisation as a fine and delicate instrument, has been accompanied by increasing development of brain power.

Stereoscopic Vision.

Although binocular vision is met with in many birds and a few fishes, and some amphibia and reptiles, yet none of these possess true stereoscopic vision, as is met with in the higher mammals, in which visual impressions from the two eyes are received on the same side of the brain, owing to the semi-decussation of the optic nerves at the chiasma. In a few mammals, as mice and other rodents, and in all the lower vertebrates, birds, reptiles, amphibia, and fishes, the decussation of the optic nerves is total, each optic nerve ending in the optic lobe of the other side. Visual impressions are further conducted to the cerebral hemisphere on the same side as the optic lobe which receives the impressions, for it has been shown in pigeons that such experimental extirpation of one cerebral hemisphere has been followed by complete blindness of the opposite eye; that is to say, psychical blindness, in which there is no definite conscious perception, though it is true that when both hemispheres have been removed it is clear that the pigeon is guided to a certain extent by reflex retinal impressions, not recognising food or the presence of an enemy, except so far as they are objects which reflect light, and so stimulate the retina. Edinger (6) states that in some birds, as the owl, the decussation of the optic nerves is only partial, but with this statement I cannot agree. Whether the opposite eye would be equally blind in an owl, which has fair binocular vision, as it has been shown to be in a pigeon, which has laterally placed eyes, after extir-

pation of the cerebral hemisphere on one side, has not to my knowledge been shown. The probability is that it would be the same as in a pigeon, since the decussation of the optic nerves seems to be as complete, and there is no known crossed afferent tract connecting the optic lobe of one side with the opposite cerebral cortex. It is therefore probable that in the owl and other birds with partial binocular vision the association of the visual impressions received by the two eyes of the same object is effected by the commissural fibres of the corpus callosum. Now, in the primates, and in less degree in other mammals with well-developed binocular vision, such as the felidæ, there is true stereoscopic vision, owing to the visual axes being approximately parallel, so that the two maculæ are brought to bear on the same object, and owing to the semi-decussation of the optic nerves at the chiasma, by means of which the fibres from the corresponding parts of the two retinæ, carrying sensory impressions of the opposite visual field, reach the external corpus geniculatum and pulvinar of the same side, and thence the occipital cortex on the same side. That is to say, the visual impressions received on the left halves of the two retinæ are superimposed, both being received by the same part of the occipital cortex of the left side, while the visual impressions received by the right halves of the two retinæ are similarly superimposed upon the right occipital cortex. A proof of the existence of stereoscopic vision in the cat is afforded by the production of homonymous hemianopia in this animal by experimental injury of the optic tract, as I shall describe later. It is worthy of note that semi-decussation of the optic nerves is a partial return to the primitive condition in which each optic nerve arises from the same side of the brain, as seen in the cyclostoma, and in the early stage of development of the mammalian eye.

Macular Vision.

According to Lindsay Johnson (12) a macula is only to be found in man and the true monkeys, which, he says, alone possess the power of convergence. He states: "The

divergence of the optic axes follows the classification to a marked degree. The higher the order the nearer the axes approach parallel vision. Parallel vision with the power of convergence only occurs in those animals which possess a true macula, viz., man and all the simiæ." Although this author recognises that periscopic vision (divergent optic axes, as in the hare and rabbit), combined with a large cornea, is a great source of protection to the animals by the increased range of vision it gives them, yet he does not see the obvious complement to this argument, that binocular vision, with less and less divergence of the optic axes, is of increasing value to predatory animals, birds as well as mammals, which have to follow instantly with their eyes the dodging movements of their prey in the endeavour to escape their pursuer. I have no doubt the macular region of the retina becomes more and more specialised in animals with increasingly binocular stereoscopic vision, and that the felidæ have no area of intense vision for fixation corresponding to the human macula I cannot accept on ophthalmoscopic examination alone, as Johnson asserts.

As regards the cortical representation of the macular region, there are two conflicting theories. The fact that, in most patients suffering from permanent hemianopia, the dividing line between the blind and the seeing halves of the field is found to pass around the fixation point, leaving it included in the seeing half of the field for each eye, has led Wilbrand (18) to suggest that there is a special bifurcation of the macular fibres at the chiasma, by which means the whole of each macula is represented in the occipital cortex on each side. He admits, however, that in many cases of hemianopia the dividing line passes directly through the fixation point, and he explains these and others in which it passes closer than usual to the fixation point, by suggesting that they are to be accounted for by individual variations in the amount of bifurcation of the macular fibres. Wilbrand and Saenger (19) quote, in support of this theory, the observations of Ramón y Cajal on cat embryos, who, using the Golgi method, has described fibres in the chiasma bifurcating, the divisions passing into the two optic tracts, though there is no evidence

that these bifurcating fibres were macular fibres. Since Cajal has also described similar bifurcation of fibres in rabbit (4), the probability of these fibres being macular fibres is very small. On the other hand, Förster (7) regards the representation of the macula on the cortex as on the same plan as the rest of the retina, so that in a total destruction of one optic tract or of the optic radiations or occipital cortex on one side, permanent hemianopia would result with the dividing line between the seeing and the blind fields passing through the fixation point. He explains the retention of a small area of vision around the fixation point in most cases of hemianopia by supposing the macular area of the cortex to have greater resisting power to embolic or thrombotic softening through better blood supply through anastomotic branches. Against this theory is the statement that in cases of destruction of one optic tract the dividing line has been found to pass around the fixation point, leaving it in the seeing half. Henschen (11), although supporting Wilbrand's theory of bifurcation of the macular fibres, considers that Förster's supposition as to the greater resistant and recuperative power of the macular area through better blood supply as very probable. Three pieces of evidence which, in my opinion, prove the truth of Förster's theory are (1) the mode of improvement of cases of hemianopia, especially of cases of transient hemianopia not due to any gross lesion of the half-vision centre around the calcarine fissure, but to exhaustion of the centre from the spread of an epileptiform discharge from a neighbouring patch of softening, such as occurred in the two cases which I published (9) of softening in the quadrate lobule, just in front of the cuneus. In such cases the visual field invariably clears up from the centre towards the periphery, the macular vision being the first to be regained, showing the greater recuperative power of the cortex associated with the representation of macular vision; (2) cases of double hemianopia have been recorded due to thrombotic softening in both cunei, in which permanent blindness did not result, but a small area of central vision returned, with diminished visual acuity, resembling in size the pin-point fields of hysteria.

Such recovery of central vision is a strong argument in support of the theory that the cortical area of representation of the macula has greater recuperative or resistant power than the rest of the half-vision centre. (3) The most convincing proof of the macular region being represented in the cortex on the same plan as the rest of the retina, namely, that half of each macula is represented in each cortex, is afforded by the study of cases of transient hemianopia. In the paper referred to above, I published eleven such studies of transient hemianopia occurring in four patients after epileptiform fits, in each of which the hemianopia was absolute for several hours, a bright flame being invisible in the blind field, which in each case extended up to the fixation point, the dividing line passing through the fixation point, so that the flame of a match or candle was not seen until it crossed that line. I have since then observed three similar cases, and in each vision began to clear up after several hours, until after twenty-four hours or a little longer the visual fields were again normal, the improvement taking place always from the centre towards the periphery. Moreover, every now and then one finds a case of permanent complete hemianopia in which the dividing line passes through the fixation point, and I have now under observation such a case in which a small flame is not seen at all to the right by the right eye until it crosses the line of fixation, though the left eye, which has only the smaller nasal field blind, sees the flame about five degrees to the right of the fixation line. This point has repeatedly been tested and confirmed by two other observers. To test this point it is not sufficiently accurate to use a perimeter, because it is then always uncertain whether the patient's eye does not waver a little. To make certain of the line of fixation, it is far more accurate to test these cases by Gowers' method (9), in which the patient sits or stands opposite the observer, each gazing into the pupil of the eye opposite, so that the line of fixation of observer and patient is the same, and the slightest wavering of the patient's eye is at once detected, and the test again commenced. Using this method in cases of transient hemianopia, I have in every case found the dividing line to pass exactly through

the fixation point, which is, in my opinion, complete proof that in all the patients examined (seven in number) there was no special bifurcation of the macular fibres.

Förster considered that the macula was represented only in the posterior part of the calcarine fissure, while Barker (1) suggests that the representation of the macular region is shared by the whole of the calcarine cortex. von Monakow (15) also considers that there is no special bifurcation of the macular fibres, and explains the persistence in most cases of hemianopia of an area of vision around the fixation point by supposing that the whole of the visual area of the cortex, occipital and angular gyrus, is connected with the macula by means of the macular fibres in the optic tract making connections with all the nerve cells in the external corpus geniculatum. This theory is, however, negatived by Henschen's clinical and pathological work on lesions of the external corpus geniculatum, in which he proved the upper half of this nucleus to correspond with the upper quadrant of the retina. An additional and conclusive fact proving the limitation of the macular area of the cortex is the occurrence of central scotomata of hemianopic type, corresponding more or less to the macular area of vision, such as Wilbrand's case, in which local lesions of the calcarine cortex have been found.

Ocular Movements, Conjugate and Independent.

In man and a few of the higher mammals with binocular stereoscopic vision, the movements of the eyes are perfectly co-ordinated, so that the eyes move conjugately in all directions, laterally and vertically, as well as in convergence. This is, as is well known, due to the impressions received from the maculæ of the two eyes being received as one object on the visual cortex, and thus reflexly through the motor action of the occipital cortex upon the oculo-motor lower centres, the two eyes are kept in conjugate movement fixed upon the object, so that diplopia does not result. This conjugate movement of the two eyes, by which the optic axes are kept trained upon the same object, is the result of long-continued education of the cortex and

the oculo-motor apparatus, for if the ocular movements of a young infant be watched, before the child has learnt to use its eyes in fixing objects, the eyes are often seen to diverge or to squint inwards, an independent action of the eyes which is also seen in older persons when one eye has developed blindness in early infancy. This conjugate movement of the two eyes for lateral and vertical movements (not for convergence), being the result of stereoscopic vision, is therefore directly associated with semi-decussation of the optic nerves at the chiasma. I have spent a considerable time studying the movements of the eyes of different animals, especially at the Zoological Gardens, with regard to the amount of conjugate or independent movement of the eyes possessed by them. It is not an easy matter with the majority of the animals to observe both eyes so as to be sure of their movements, especially when the eyes are placed more or less laterally, as in the sheep, oxen, and other ungulata. In the rodents I have not been able to observe any definite ocular movements at all. In monkeys and in the larger cats among the carnivora it is easy to see well-marked conjugate movements of the eyes resembling those of man, and their eyes are similarly placed in the head, with good stereoscopic vision, and I have found in sections of the chiasma of a cat stained with Marchi's method, after enucleation of one eye a fortnight previously, that a large proportion of degenerated fibres were present in the optic tract of the same side (fig. 8). In bears, a group of the carnivora which is mainly frugivorous, there is not nearly such perfect binocular vision as in the cats, the eyes at rest being divergent, and occasionally distinct independent movements of the two eyes can be seen, especially in the vertical direction. Yet the majority of the movements are conjugate, laterally and vertically, and the eyes can be brought forwards more or less in convergence. Of the ungulata, I have studied the ocular movements in the horse, ox, deer, antelopes, and sheep (mouflon and thar). In all these forms the eyes are set much more laterally than in the bear, and binocular vision is necessarily defective in these animals. Attempts at convergence are seen in all of them in the effort

to look at an object directly in front of them, but it is of course imperfect. There is also a certain amount of lateral conjugate movement, but independent movements of the eyes are more easily seen in the ungulata, and are more frequent than in the bear, and occur both in lateral and vertical directions. This greater independence of movement of the two eyes is associated with a less complete stereoscopic vision, the decussation of the optic nerves being more nearly total in these animals, in the horse about one-

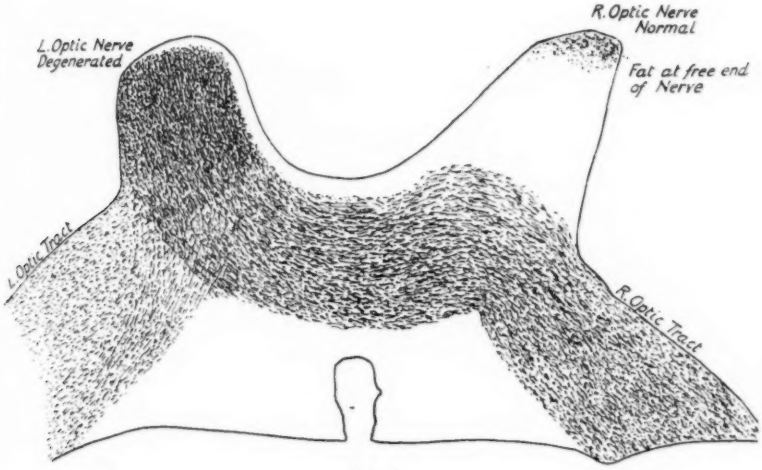

FIG. 8.

Section of chiasma of cat, stained by Marchi's method.

sixth of the fibres of one optic nerve being said to pass into the optic tract of the same side. In rodents, such as rabbit and hare, conjugate movements of the two eyes could serve no useful purpose, and are practically impossible, since the eyes are set almost completely laterally, with next to no power of binocular vision. In these animals the decussation of the optic nerves is practically complete. Using Marchi's method after enucleation of one eye in a rabbit a fortnight previously to staining, I found only a very few fibres here

and there turning round from the optic nerve into the optic tract of the same side, the vast majority decussating into the opposite optic tract (fig. 7). This is also figured by Ramón y Cajal (4). As I have previously said, in all animals below mammals, whether possessed of binocular vision or not, the decussation of the optic nerves at the chiasma is complete, and they have therefore no stereoscopic vision.

Yet in many of them there is fair power of convergence of the eyes, though used solely, as far as I have been able to observe, in feeding. As I have already shown, the chameleon has the most perfect power of convergence of the eyes (see fig. 4) in the act of focussing its prey before striking with the tongue, while at all other times its eyes show the most complete independence of movement over a very wide range (see fig. 3). I have watched a large number of birds of different kinds, but in the majority it is difficult to make out any definite ocular movements, any change of direction of vision being performed by movements of the head. Especially is this the case with the owls, in none of which could I observe a trace of ocular movement. Indeed, the shape of this bird's eye is ill adapted for movements within the orbit, being somewhat egg-shaped, with a constriction round the smaller end behind the cornea. The eye is deep set, and the cornea fills the entire ocular fissure, so that very slight movements would not be easily seen. In the hornbills, however, the eyes, though much more laterally placed, are more prominent on the side of the head, and the eye movements are easily studied. The movements of the two eyes, as in the chameleon, are perfectly independent for lateral and vertical movements, though in taking food both eyes are brought forwards to bear on the object in distinct convergence. Since the eyes are, however, not so freely movable as in the chameleon, the convergence is not so perfect. The large concave-casqued hornbill at the Zoological Gardens, *Dichoceros bicornis*, makes use of this power of convergence in catching grapes or nuts thrown to it with wonderful precision.

It is thus clear that though the power of convergence is

met with in animals with independent movement of the eyes and periscopic vision, yet the acquirement of conjugate movement of the eyes for lateral and vertical movements is directly dependent on the acquirement of stereoscopic vision, and the perfection of conjugate movement and the absence of independent movements of the eyes is directly in proportion to the extent of the stereoscopic vision, which is in turn dependent upon the forward setting of the eyes in the head, with parallel visual axes, and semi-decussation of the optic nerves at the chiasma. Thus the most perfect conjugate movements of the eyes, with almost complete absence of any independent movement, is seen in man and monkeys, whose eyes are set parallel, with semi-decussation of the optic nerves, and good stereoscopic vision, while complete independence of the eyes in lateral and vertical movements is seen in animals with laterally placed eyes, total decussation of the optic nerves, and periscopic vision, such as the chameleon and the hornbill. The stereoscopic vision of man, though the highest development, is not ideally perfect, since, owing to the projection of the nose, the nasal field is much smaller than the temporal, and the uncrossed bundle in the optic tract is correspondingly smaller than that which has decussated from the opposite eye.

The Path of the Pupil Reflex to Light.

There can be no doubt that the pupil reflex fibres for light leave the retina by the optic nerve, and run with the visual fibres in the nerve at least as far as the optic chiasma, since severance of the optic nerve, or destruction of the chiasma from the pressure of a tumour, cause abolition of the pupil reflex in addition to blindness. Occasionally exceptional cases are seen in which, although there is no perception of light from retinal disease or optic atrophy, yet the pupil may react fairly readily to light. Such are probably to be explained by supposing that the pupil reflex fibres are less easily damaged than the visual fibres. Beyond the chiasma various courses have been allotted to them. According to Henschen, the pupil fibres form a small bundle

on the dorso-medial aspect of the tract. von Bechterew (2) states that they leave the chiasma posteriorly, and without decussating penetrate the floor of the third ventricle, passing uncrossed to reach the third nucleus. One of his reasons for this view is that after experimental section of the optic tract in the dog, he produced hemianopia but no alteration of the pupil. Now the dog is not nearly such a good animal for this experiment as the cat, which has a greater amount of binocular vision, keener sight, and a much more mobile pupil, reacting as briskly to light as the human pupil, while that of the dog at best reacts sluggishly. I have repeated Bechterew's experiment in cats, and have found in five animals well-marked hemianopia with the hemianopic pupil reaction in two of them, and sluggishness of the opposite pupil in all five, after injury to the optic tract on one side. Again, Darkschewitsch (5) describes the pupil reflex fibres as leaving the optic tract near the external geniculate body, and passing through the thalamus to the ganglion habenulæ and the pineal body, decussating in the posterior commissure with those of the other side, to reach the upper oculo-motor nucleus. In none of my five experiments in which the optic tract was injured did I find any degenerated fibres by Marchi's method passing into the ganglion habenulæ, or decussating in the posterior commissure; while in two other experiments in which I divided the posterior commissure, although there was temporary loss of pupil reaction (probably from shock), there was no permanent effect whatever on the pupils.

These experiments, and those necessitating the enucleation of one eye in pigeon, owl, rabbit, and cat, were performed, under the usual licences, in the Research Laboratory of Professor Vaughan Harley at University College, part of the cost being defrayed by a grant from the Scientific Grants Committee of the British Medical Association.

Description of Experimental Procedure for Dividing One Optic Tract.

In each case the cat was anæsthetised with ether, and anæsthesia kept up until the conclusion of the operation,

when, after suturing and dressing the wound with wool and collodion, the animals were kept in separate cages, and studied at intervals during three weeks. The animals were then killed, after a final examination, and the brains at once taken out and placed in Müller's fluid for microscopical examination.

Operation.—The top of the head was shaved, washed with soap, ether and turpentine, and lastly with 1 in 1,000 perchloride of mercury solution. A semicircular flap of skin was then reflected over the left ear, the temporal muscle separated from the bone and cleared away from behind forwards, and the root of the zygoma exposed and carefully cleaned. A quarter of an inch trephine hole was then made immediately above the root of the zygoma, and the hole enlarged by bone forceps upwards, forwards, and backwards, to the size of a halfpenny. The dura mater was then incised around the lower three-quarters of this opening, and the flap reflected upwards. This exposed the posterior ecto-sylvian fissure running vertically across the middle of the trephine opening, opposite the root of the zygoma. Through the middle of this fissure I then plunged horizontally a blunt-pointed Graefe's knife to the depth of three-quarters of an inch, cutting with the point slightly forwards. The dura was then replaced, the muscle flap returned, and the skin wound sewn up by several silk stitches, covered with boracic powder, and then with cotton wool and collodion, which was allowed to dry. The dressing was generally torn off by the animals within half an hour of their recovery from the anæsthetic, but the wounds mostly did well in spite of this.

Cat 1.—Male. Immediately after the operation the right pupil was found to be dilated and very sluggish in its reaction to a strong light held in front of it, while the left pupil reacted more briskly on exposure to light, with consensual reaction of the right pupil. On focussing the light so as to illuminate the two halves of the left retina alternately, it was found that the pupil only reacted when light was thrown on it from the left side, that is to say, the hemianopic pupil reaction could be demonstrated in the left eye. Five days later, the condition of the pupils was

just as before, while there was marked right hemianopia. Some weakness of the hind limbs was apparent, and some forced movements of the trunk to the left. Three weeks after the operation the forced movements to the left had disappeared, and the hind limbs had quite recovered. The right pupil was still larger than the left, and reacted sluggishly to a bright light held directly in front, or on the left side, the left eye being shaded. No reaction of the right pupil to light thrown on it from the right side. The left pupil then reacted briskly to light thrown on it directly from the front or from the left side, with well-marked consensual contraction of the right pupil. No contraction of either pupil when light thrown on the left pupil from the right side. Both pupils contract and dilate consensually as the animal moves its eyes about, and when its attention is engaged. Examination of the animal's vision showed typical hemianopia on the right side. Each eye was in turn obscured by strapping, and it was found that the animal showed no sign of flinching or palpebral reflex to a finger darted at the eye from the right side, though on darting the finger at the left eye from the left side there was most marked flinching. Vision was certainly defective in the whole of the right eye, as was suggested by the sluggishness of the right pupil to light thrown directly on it, and the animal did not flinch at all when the finger was darted at the right eye from either direction. Yet the animal could see with this eye, for with the left eye covered with strapping, the animal could see sufficiently to find its way about, running up to the window and stopping short of the glass quite naturally, and going up to the leg of the table to try and rub off the strapping. The animal was then killed with an overdose of chloroform, and while under the anæsthetic the fundi of both eyes were examined with the ophthalmoscope, no difference in either being seen from a normal cat's fundus. The autopsy showed that the left optic tract had been wounded half an inch above the chiasma, about the anterior half of its fibres having been cut across.

Cat 2.—Medium sized male. Usual operation. Immediately afterwards the right pupil was larger than the left, and did not react directly to light, but did so consensually when light was thrown on the left pupil. The left pupil reacted briskly. The next day there was marked right hemianopia, with slight right-sided weakness and right fore-foot drop. Three weeks later there was still marked forced movement to the left. Well-marked right hemianopia, right pupil > left. No hemianopic pupil reaction could be demonstrated in this animal, though the

right pupil reacted sluggishly to direct light, and more briskly consensually when the left pupil was exposed to light. At the autopsy the left optic tract was found completely cut across.

Cat 3.—Small female. Usual operation. The fissures were seen to differ somewhat from the ordinary, the posterior ecto-sylvian being short and slanting rather backwards. There were no forced movements after this operation, the animal running about quite straight an hour afterwards. There was obvious right hemianopia, the right pupil being > left, and with sluggish reaction to direct light, but reacting briskly consensually when the left pupil was exposed to light. Twenty-seven days later before killing the animal, the notes are as follows: "In good health, obvious right hemianopia, each eye tested separately, vision evidently commencing in mid line for each eye. Vision in right eye less acute than in left. Right pupil > left, and reacts sluggishly to direct light, but briskly consensually when left pupil is exposed to light. Left pupil reacts much more briskly when light is thrown on the right side of the retina than on the left side, but there is distinct reaction in the latter case also. The same, though less, for the right pupil. After killing the animal with an overdose of chloroform nine weeks after the operation and taking out the brain, the left optic tract was found wounded a quarter of an inch below the external geniculate body, but it had not been completely cut through. There was marked atrophy of the left external geniculate body and the portion of the optic tract between it and the scar, the left external geniculate body being about a quarter the size of the right one. There was no atrophy of the anterior corpus quadrigeminum, or of the pineal striæ, or of the lower part of the optic tract or chiasma.

Cat 4.—Small female. Usual operation. Immediately afterwards the right pupil was slightly larger than the left, and reacted very sluggishly to direct light, but fairly consensually. There was obvious right hemianopia, and slight right fore-foot drop. Seven weeks later the right hemianopia still persisted, the right pupil was still slightly the larger, and reacted more briskly consensually than to direct exposure to light. There was, however, no difference in the reaction to be demonstrated on focussing the light from either side on either of the pupils. At the autopsy a wound was found in the left optic tract at the neck of the external corpus geniculatum, the anterior half being divided.

Cat 5.—Medium sized female. Usual operation. The posterior ecto-sylvian fissure, which I always used as my guide for plunging in the knife to reach the tract, was in this case slightly in front of the root of the zygoma. For several hours after the operation there was marked right-sided weakness, and forced movements to the left, the animal running round after its tail. No nystagmus. Right pupil > left, with no direct reaction to light, but fair reaction consensually. The left pupil reacted fairly, and though there was obvious right hemianopia, no hemianopic pupil reaction could be made out. Nine weeks later, the hemianopia persisted, no flinching to a finger darted from the right side, though flinching at once to a finger darted at the eyes from the left side. The right pupil was very slightly > the left pupil, and reacted not quite so briskly to direct light. At the autopsy the left optic tract was found wounded at the neck of the external corpus geniculatum, the anterior half of the tract having been cut through.

Summary of Results of Wounding the Optic Tract.

Persistent hemianopia resulted in each of the cases in which the optic tract was wounded, in only one of the five cases the tract being completely divided. In each case the vision of the eye on the side opposite to that operated on was less acute than that of the eye on the same side, while the pupil reaction of the crossed eye was much more sluggish to direct light than that of the eye on the same side, and at the same time the consensual pupil contraction of the crossed eye was in each case brisker than its reaction to direct exposure to light. In only two of the five cases could the hemianopic pupil reaction be made out, in Nos. 1 and 3, three weeks and four weeks after the operation. In the first the tract was only half cut through, and in the other the tract was not quite completely cut through. No special apparatus was employed in testing for the hemianopic pupil, the light of a lamp only being focussed on the pupil from one side by means of a lens. Possibly owing to this imperfect method of confining the beam of light to one side of the retina was due my failure to obtain the hemianopic pupil reaction in every case, though it is noteworthy that in the only case, No 2, in which the tract was found to

have been completely cut across, the pupils appeared to react equally well when the light was thrown from either side, although obvious hemianopia was present. Boyce (3), who divided the left optic tract in his operation for removing the hemisphere, states that this was followed by complete blindness in the right eye and slight constriction of the field in the left eye, but, as I have shown, my results do not agree with this. On examining my sections of a cat's chiasma, stained with Marchi's method (fig. 8) after enucleation of one eye three weeks previously, the number of the decussating is clearly greater than that of the uncrossed fibres. The figure of the cat's chiasma in Ramón y Cajal's work (4) shows the same outward sweep of the decussating fibres towards the root of the healthy optic nerve, as I found in my sections, and shows quite twice the number of decussating as of uncrossed fibres, which, indeed, I should have expected from the condition of the cat's vision and of its pupils in each of my five successful cases after wounding the optic tract.

It was clear from my results in these animals that the decussation of the optic nerves must be more complete in the cat than in man, less fibres turning into the tract of the same side, since the vision of the opposite eye was more affected in every case, and the pupil reaction of that eye was much more sluggish to direct exposure to light than its consensual reaction when the pupil on the same side was exposed to light. Stereoscopic vision in the cat, therefore, is less perfect than in man, in whom the vision of the two eyes in homonymous hemianopia is equally acute, and the pupil reaction to light in optic tract lesions about equally brisk in both.

In order to test the proof of Darkschewitsch's theory that the pupil reflex fibres decussate in the posterior commissure, I made four attempts to divide this commissure in cats, two of which were successful. In each case immediately after the operation the pupils were widely dilated and fixed, due, I think, largely to the greater severity of the operation. In three of the cases, however, the pupils recovered their normal movements, and in two of them at

the autopsy the posterior commissure was found to have been successfully divided without injuring the corpora quadrigemina, while in another animal in which the incision missed the posterior commissure a deep cut was found in the right anterior corpus quadrigeminum close to the mid line, extending deeply to the floor of the aqueduct, and passing forwards and slightly outwards towards the inner part of the right external corpus geniculatum. In this animal, though the animal recovered sufficiently from the anæsthetic to growl, and the palpebral reflexes returned, yet the pupils remained widely dilated, with no trace of reaction to light. Unfortunately the animal died six hours later.

My operative procedure in these cases was as follows:—

Operation.—After shaving the head, and the usual antiseptic precautions, the animal being anæsthetised by ether, a longitudinal incision 2½ inches long was made in the median line from the level of the ears. After reflecting the skin and temporal muscle on the left side, I then made a small trephine opening in the parietal eminence on the left side, and then cut away the bone with forceps up to within a quarter of an inch of the median line, after changing the anæsthetic to chloroform on account of the free bleeding from the bone, which was stopped by plugging it with aseptic beeswax. The bone was then, after waiting a few minutes, cut away across the mid line and down on the right side, and was taken away posteriorly as far as the junction with the bony tentorium, leaving a hole about half an inch in length and 1½ inches in width across the middle line of the vertex. The dura was next incised along the posterior margin of the opening on the left side and reflected forwards, the posterior portion of the left hemisphere then being retracted to the left by an aneurysm needle inserted under the dura. A canaliculus knife was then passed downwards vertically in the middle line, the notch in the bony tentorium being the guide to the middle line, and being passed down to a distance previously measured on the knife as calculated to just pass the posterior commissure. The knife was drawn forwards with a cutting motion for about half an inch, the posterior margin of the corpus

callosum thus being incised for about one-third of an inch, as well as the posterior commissure, which lies more deeply beneath it. The wound was then sewn up with separate silk ligatures dressed in the usual way, and the animal allowed to recover.

Of four attempts I made in this way to divide the posterior commissure only two were successful, without injuring the corpus quadrigeminum. In all the cases there was complete loss of pupil reaction to light immediately after the operation, remaining in one case for as long as three days, though the pupils still contracted and dilated consensually with the movements of the eyes. In all the animals which survived, however, the pupil reactions returned, becoming quite normal, including the two successful cases in which at the autopsy the posterior commissure was found cut, with no wound in the anterior corpus quadrigeminum of either side.

Although it appears that this operation is a greater shock to the pupil-reflex centres than is the division of one optic tract, yet the recovery of the pupil reactions to light after division of the posterior commissure disposes of Darkschewitsch's theory of the pupil fibres entering the ganglion habenulæ and pineal body, and decussating in the posterior commissure. It is indeed practically certain that the pupil reflex fibres, probably the fine nerve fibres contained in the optic nerve, reach nerve cells in the superficial grey matter of the anterior corpus quadrigeminum. On tracing the degenerated nerve fibres by Marchi's method in those cats in which I had successfully injured the optic tract, these fibres were found to surround the external geniculate body, and then by the anterior brachium to spread out over the surface of the anterior corpus quadrigeminum, none of the fibres passing into the ganglion habenulæ or into the posterior commissure (fig. 9). Indeed, it was obvious that the fibres reaching the corpus quadrigeminum by the anterior brachium must end in the grey matter of this body. Whether any of these degenerated fibres are pupil reflex fibres it is impossible to say. Probably the majority of them are fibres subserving a reflex function connecting

vision with the ocular movements, especially the reflex, by means of which the eyes are held steady in position when the two eyes are directed attentively towards an object. It is possible that the fibres concerned in the pupil reflex to light running in the optic nerves do not degenerate centrally after division of the nerve or tract, if it is true that these fibres are the fine nerve fibres running peripherally to the retina, having their cells of origin in the corpus quadrigeminum, as Ramón y Cayal described.

In four of my animals (cats), in which I accidentally injured the anterior corpus quadrigeminum, I was able to

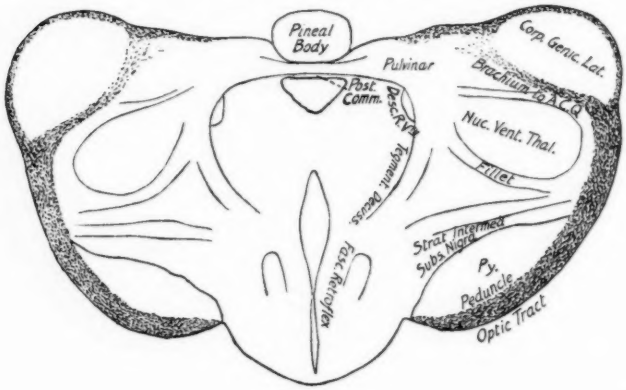

FIG. 9.

Section at level of posterior commissure of cat, stained by Marchi's method, after enucleation of left eye.

trace degenerated fibres starting from the central grey matter surrounding the aqueduct immediately beneath the wound in the anterior corpus quadrigeminum, and sweeping round ventrally beneath the third nerve nucleus and the posterior longitudinal bundle, the majority decussating in the raphé and then turning caudally to form a tract running longitudinally to end in the anterior columnar fibres of the cord, as Boyce described. This is the tract named by Forel the fountain-like tegmental decussation of Meynert. This was the only degeneration I found resulting from these localised

lesions of the outer and dorsal portion of the anterior corpus quadrigeminum, and it seems to me an attractive theory, as I stated in my paper before the Neurological Society in December, 1899, that these fibres are the relay continuing the path for the pupil reflex to light to the third nuclei, which has been interrupted by the nerve cells in the superficial grey matter of the corpus quadrigeminum. Not all the fibres in Meynert's fountain decussation in the cat decussate, as Boyce stated, and as I showed in my sections also, a very few running caudally in the posterior longitudinal bundle of the same side, and it seems not improbable, as Held suggested, that these fibres assist in connecting the visual reflex with all the oculo-motor nuclei and with the lower centres in the medulla and neck for turning the head.[1]

I have studied the pupil reflex to light in a considerable number of animals, frog, toad, pigeon, parrot, owl, rabbit, horse, dog, cat, monkey, and in man, and I have found that in all those animals in which there is a total decussation of the optic nerves at the chiasma, only that pupil contracts which is exposed to light. The toad's pupil is oval horizontally, and reacts very sluggishly to light, but still very definite contraction takes place, and only in the pupil exposed to the light. It is said that in these animals the iris is directly sensitive to light, but in the higher animals this is certainly not so. I have cut sections of the chiasma of a toad, stained with Weigert-Pal, and found complete decussation of the optic nerve fibres. I have also cut similar sections of the chiasma of the chameleon, and found the same total decussation of the fibres (fig. 5), but the pupil of the chameleon is too small and deeply set for any observation of its reflex to light. Considerable care is necessary in studying the pupil reflex in birds, as they possess voluntary power of constricting the pupil, in addition to its reflex contractility, the sphincter iridis in birds containing striped muscular

[1] This *fontainartige Haubenkreuzung* of Meynert must be distinguished from the "superior commissure of Meynert," which lies dorsal to Gudden's commissure, beneath the third ventricle, and also from another bundle known as "Meynert's bundle," which connects the ganglion habenulæ with the opposite interpeduncular ganglion. This latter bundle is sometimes known as the *fasciculus retroflexus*, and has no connection with the pupil light reflex.

fibres, and being unaffected by atropine. This voluntary constriction of the pupil I have noticed more in the grey African parrot than in pigeons or owls. In the parrot or pigeon, owing to the lateral position of the eyes, it is impossible to watch both pupils at the same moment, and I therefore adopted the method of fixing a mirror near one side of the bird's head, and watching the reflexion of the pupil on that side in the mirror, while throwing a beam of light upon the other pupil. After several trials, the bird becomes accustomed to the flashing of the light, and no longer contracts its pupils voluntarily at the moment of the flash. This voluntary sphincter action of the pupil is consensual, both pupils contracting equally, while the reflex contraction of the pupil to light is unilateral, only the pupil on which light is thrown contracting. The same unilateral contraction of the pupil is found in owls, although they have considerable binocular vision, and the decussation of the optic nerves in the owl, as in the pigeon, is total. I have traced the degenerated fibres in the pigeon, a fortnight after enucleation of one eye, and then cutting sections of the chiasma and optic lobes, stained by Marchi's method. The decussation is then seen to be total, all the degenerated fibres passing into the short optic tract of the other side, passing external to the insignificant geniculate ganglion, and spreading out over the surface of the optic lobe. A small bundle, the "median optic bundle," is seen running dorsally near the posterior commissure, as described by Wallenberg, and by Boyce and Warrington. This bundle has been shown by Wallenberg and Edinger to contain centrifugal fibres which degenerate downwards from the ganglion isthmi to the retina. Possibly these are the pupil fibres. There is no direct connection between the optic fibres and the third nucleus, no degenerated fibres being traced beyond the optic lobe, which corresponds to the anterior corpus quadrigeminum and pulvinar in mammals. Since, therefore, only that pupil contracts which is exposed to light, and the decussation of the optic nerves is total, it necessarily follows that there must also be a further posterior total decussation of the pupil reflex fibres between the grey matter of the optic lobes

and the sphincter centre in the third nerve nucleus. Moreover, the two sphincter centres of the two third-nerve nuclei cannot be tied together by commissural fibres. The accompanying diagram fig. (10) illustrates what must of necessity be the approximate course of the pupil reflex fibres in birds and lower animals with total decussation of the optic nerves. In some mammals, such as mice, rats, and other rodents, the optic nerve decussation is either total or practically so, as I have shown to be the case for rabbits, and I have found the

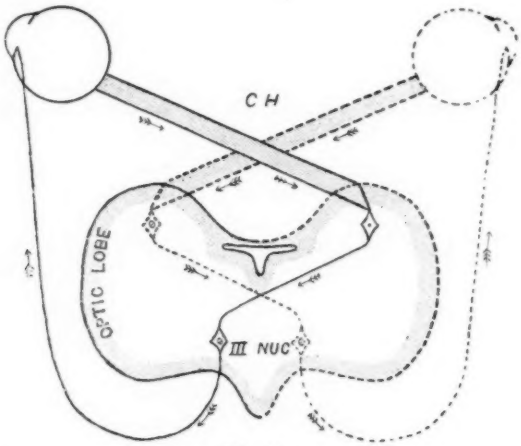

FIG. 10.

Diagram of the pupil reflex to light in birds and reptiles, and some of the lower mammals.

pupil reflex to light in rabbits to behave exactly as in the bird or toad, only that pupil contracting on which light is thrown. The rabbit's pupil is sluggish in reaction, this sluggishness probably corresponding with a poor acuity of vision. Indeed, sluggishness of pupil reaction seems to me to be associated generally with a low standard of acuteness of vision, as in ungulata and rodents, while in birds, some carnivora, as the cats, monkeys, and man, with a briskly reacting pupil to light, sharpness of vision is correspondingly more developed.

Amongst the carnivora, the cat's pupil reacts much more

briskly than that of the dog, the former animal watching for its prey in dim light, which necessitates keen vision, while the latter is known to depend more upon the sense of smell than on that of sight. In the horse the pupil reacts sluggishly, as in the rabbit, and the contraction is much greater in the eye exposed to light than in the shaded one, though there does appear to be a slight amount of consensual pupil reaction, as one would expect, on account of the partial decussation of the optic nerves, one-sixth of the fibres being said to pass into the optic tract of the same side. The dog shows very fair consensual reaction of the pupils when one is exposed to light, and the same is seen in the cat. That this mechanism for the consensual contraction of the pupils is not nearly so well developed in the cat as in man is well brought out by my results on the pupil reactions after injury to the optic tract on one side. As I have already stated, after injury to the optic tract in five cats, in each case I found, although there was clearly hemianopia, yet the pupil of the opposite eye was larger, and reacted much more sluggishly on direct exposure to light than its consensual reaction when the pupil on the same side as the injury was stimulated. This indicates that the decussating optic bundle is much larger than the uncrossed bundle in the cat, as Ramón y Cajal has figured, and therefore the light stimulus, when conducted only by the small uncrossed bundle from the opposite eye, is much more feeble in causing pupil contraction than when it is conducted by the large decussating bundle from the nasal side of the retina on the same side as the injury. I have not attempted to divide the optic tract on one side in monkeys, but in all probability the results would be somewhat similar to that seen in man in cases of diseases of the optic tract in which the hemianopic pupil reaction of Wernicke is seen, in which cases the briskness of reaction of the two pupils is nearly equal, whichever pupil is exposed to light. The size of the uncrossed bundle in man, and probably in monkeys, is proportionately larger than in cats, being more nearly equal in size to the decussating bundle, and its proportion is stated by Barker to vary probably from 1:2 to

3:5. Although human cases are on record (10) in which the whole optic nerve of one side was found to pass direct into the optic tract of the other side, yet the views of von Kölliker, though expressed so recently as 1896 (13), that the optic nerves in man, dog, cat, &c., undergo complete decussation in the chiasma, are now only of historical interest, the results of tracing degenerations by Marchi's method being absolutely conclusive to the contrary, at any rate for man and the higher mammals. Since, therefore, unilateral pupil

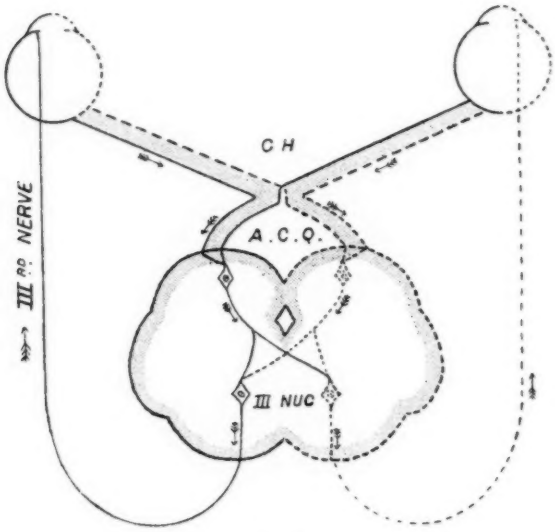

FIG. 11.

Diagram of the pupil reflex to light in man and the higher mammals.

reaction to light (birds, amphibia, rodents) is associated with total decussation of the optic nerves, so consensual pupil reaction to light is associated with partial decussation at the chiasma. In the former, in order to explain the fact that only the pupil which is exposed to light contracts, although there is total crossing of the optic nerves, there must exist a posterior total decussation of the pupil reflex fibres between

the optic lobes and the third nuclei, which cannot be tied together by commissural fibres, at least for the two sphincter pupillæ centres (see fig. 10). To explain the consensual reaction of the pupils in man, cat, &c., it is then more consistent to suppose that the posterior decussation of the pupil reflex fibres is now partial instead of total, in the same proportion as the decussation at the chiasma has changed from total to partial crossing of the fibres (see fig. 11). This seems much more rational than to explain the consensual reaction of the two pupils by introducing a new type of fibre, viz., a commissural bundle of fibres tying together the two sphincter pupillæ centres, a commissure which we have shown to be impossible in the bird and other animals with total decussation of the optic nerves, whose pupils do not contract consensually to light, though they do so to emotional reflexes, and to accommodation. That the former is the route of the consensual pupil reflex, and not *viâ* any commissural bundle uniting the two sphincter pupillæ centres, is proved by a study of the pupil reaction in cases of uniocular Argyll-Robertson pupil.

Argyll-Robertson Pupil.

It is usually accepted now, on physiological grounds, that the site of the lesion in cases of reflex iridoplegia must lie between the optic tract and the third nuclei, somewhere in the interbrain, and opinions are divided as to whether the lesion is one of sclerosis of afferent fibres connecting the anterior corpora quadrigemina with the third nuclei, or whether there is an actual nuclear degeneration. Neither theory rests on pathological data or on experimental evidence. Against the former theory has been urged the fact that Argyll-Robertson pupil is sometimes accompanied by other evidences of nuclear palsy, as paralysis of accommodation, and weakness of the external ocular muscles, while against the nuclear theory may be urged the normal action of the sphincter iridis during convergence, while it does not respond to a light stimulus, and also the extreme contraction of the pupil in many cases. If the lesion were nuclear, it is

difficult to understand how contraction, and not always dilatation, from paralysis of the sphincter centre, is produced.

The point that first led me to discard the nuclear theory was the results I obtained on testing cases of uniocular Argyll-Robertson pupil. I have now seen a considerable number of such cases, some in tabes, some in general paralysis, and several in patients showing signs of neither of these diseases, but in the large majority I have been able to obtain a distinct history of syphilis. If in such a case light be thrown on the affected pupil, although it does not contract itself, yet the sound pupil contracts consensually, a fact usually explained by the light impulses passing up the opposite optic tract to the third nucleus of the opposite side. Now, supposing for the moment that the lesion is in the third nucleus, if light be thrown obliquely on the temporal half of the affected pupil, as in testing for the hemianopic pupil reaction, then the impulses are confined to the uncrossed bundle in the optic tract of the same side, and reaching the diseased third nucleus, are stopped by the lesion, and neither pupil should contract, as it is inconceivable that the stimulus could pass through the lesion to reach the opposite third nucleus by the hypothetical commissural fibres binding together the two sphincter pupillæ centres without causing both pupils to contract. However, on performing this test carefully in every case of uniocular Argyll-Robertson pupil I have met with, there is equally brisk consensual contraction of the opposite pupil whichever half of the retina of the affected eye be illuminated. This seems to me conclusive against the theory which places the lesion of Argyll-Robertson pupil in the nucleus itself, if, as is usually figured in the text-books (17), one optic tract is connected through the corpus quadrigeminum only with the third nucleus of the same side. Now study of the mechanism of the pupil reflex to light in animals and birds proves the necessity for a posterior decussation of the pupil reflex fibres between the corpus quadrigeminum or optic lobes and the third nuclei, total for those animals with total decussation of the optic nerves at the chiasma, and partial for those animals with semi-decussation of the optic

nerves, in proportion to the size of the uncrossed bundle. Such a path is provided by Meynert's fountain decussation which has been described above, and in my opinion some of these fibres serve the function of conveying the impulses of the light reflex from the corpus quadrigeminum to the two third nuclei of the same and of the opposite sides. Although there is no positive evidence, I believe that the lesion of Argyll-Robertson pupil is a sclerosis of these fibres, especially of their terminations in the neighbourhood of the third nerve nuclei, an hypothesis which will account for all the phenomena of reflex iridoplegia. Such a sclerosis of an afferent tract is much more in harmony with the general pathology of tabes, which is essentially a degeneration of the afferent systems of neurones, rather than a nuclear degeneration would be, nuclear lesions being comparatively uncommon in this disease. In the dilated type of Argyll-Robertson pupil there is no contraction of the pupil either to light or to accommodation or convergence, and it is probable that in these cases the lesion is now affecting also the sphincter pupillæ centre in the third nucleus. In other cases the centre for accommodation is paralysed also, with varying forms of external muscular paralysis, according to the extent of the nuclear degeneration. The typical form of Argyll-Robertson pupil is minutely contracted, due no doubt to additional sclerosis of the sympathetic dilator fibres, with some additional contracture of the muscle of the sphincter iridis due to the loss of the tonic dilator stimuli of the sympathetic.

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PROCEEDINGS OF THE NEUROLOGICAL SOCIETY OF THE UNITED KINGDOM.

At a Meeting held at 11, Chandos Street, on Thursday, March 17, at 8.30 p.m., Dr. Alfred W. Campbell read a paper on "Histological Studies in Cerebral Localisation."

Abstract of Paper.

The aim of this work is to further the establishment of a correlation between physiological function and histological structure.

The normal topographic variations in arrangement of cortical nerve-cells and fibres have been relied upon in forming judgment on points bearing on localisation. A complete examination of a series of human and anthropoid apes' brains forms the basis of the research, and pathological material has been employed for confirmatory purposes.

The following areas have been defined, and will be briefly discussed:—

(1) *The "Precentral" or "Motor" Area.*

Confined to the precentral gyrus and the coterminous portion of the paracentral lobule: the "precentral" cortex is characterised by a great wealth of fibres and the presence of the "giant cells" of Betz. The same features distinguish the anthropoid "motor" area as defined by Sherrington and Grünbaum.

It has been found in two cases of amyotrophic lateral sclerosis that profound changes affect this area.

Differential localisation has been effected by the examination of seven hemispheres from cases of amputation, and by the discovery of *réaction à distance* (Marinesco) in the "giant cells" supposed to control muscles in the amputated member.

(2) "*Postcentral*" *Area.*

This area, readily defined in both man and the man-like ape, is limited to the postcentral gyrus and its paracentral annexe. The floor of the Rolandic fissure forms the dividing line between it and the "precentral" area, and its cortex differs absolutely from that of the latter.

Its supposed motor function is denied, and it is contended that it constitutes the terminus where fibres conveying common sensory impressions primarily impinge, on the following grounds: physiologically it is "silent" cortex (Sherrington and Grünbaum); its fibres myelinate early; it is the terminus for the "cortical lemniscus"; personal observations on three cases of tabes dorsalis have disclosed profound changes concentrated in the area.

(3) "*Intermediate Postcentral*" *Area.*

This is a skirting zone in which the structural type is intermediate between that of the "postcentral" area and the remaining parietal gyri.

(4) "*Parietal*" *Area.*

Roughly speaking this covers the superior parietal gyrus and the precuneus. It may share with the "intermediate postcentral" cortex the function of transmuting and elaborating sensory impressions primarily received in the "postcentral" cortex.

(5) "*Visuo-Sensory*" *and* "*Visuo-Psychic*" *Areas.*

The distribution of the "visuo-sensory" area is closely determined by the calcarine fissure, for the histologist its chief feature is the line of Gennari. It is believed to be designed for the primary reception of visual sensations, because its projection fibres myelinate prior to any others in the occipital lobe, and because clinicians agree that a minimal lesion in the calcarine region is equivalent to the production of a maximum of blindness.

The "visuo-psychic" area forms a wide, investing zone to the foregoing, and a remarkable wealth of fibres coupled with curious large pyramidal cells serve for its identification. It is argued that this cortex is specialised for the final elaboration and interpretation of sensations first received as crude impressions in the calcarine area.

The analogous areas in the anthropoid ape extend much more widely on the lateral surface of the hemisphere.

(6) "*Audito-sensory*" *and* "*Audito-psychic*" *Areas.*

These are the two specialised fields, definable in the temporal lobe. The first-named occupies the transverse temporal gyri. Clinical, developmental and anatomical reasons can be advanced in favour of the assumption that it constitutes the primary auditory centre and it has now been found to be the seat of grave changes in a case of deaf-mutism.

The "audito-psychic" area covers the posterior three-fifths of the first temporal gyrus and corresponds with the well-known centre for "word-deafness."

Similar areas have been found in the anthropoid brain.

The remainder of the temporal cortex, including the angular gyrus, is unspecialised.

(7) "*Olfactory*" *Area.*

Histology supports comparative anatomy in suggesting that the lobus pyriformis is the principal cortical centre governing the olfactory sense.

No explanation of the peculiar structure of the fissura hippocampi and gyrus dentatus is attempted.

(8) "*Limbic*" *Area.*

This practically covers the whole gyrus fornicatus; in the pregenual part cells are found resembling ones found in the lobus pyriformis, and it seems likely that these are end-stations for fibres of the inner olfactory root. The primitive cortical structure of the area in general belittles its functional importance.

(9) "*Intermediate Precentral*" *Area.*

This forms an investing zone on the anterior side of the "precentral" area. Embracing as it does the field of Broca and the supposed cheirographic centre, it is believed that it governs the execution of skilled movements. Its anterior boundary in the ape coincides with that of the hitherto accepted "motor" area.

(10) "*Frontal*" *and* "*Prefrontal*" *Areas.*

The rest of the frontal cortex is structurally divisible into two further zones of which the hinder ("frontal") is the more extensive. The feeble structural representation of the "prefrontal" cortex suggests that it is the last to appear in the course of phylogeny, and the idea is favoured that its physiological importance as a psychic centre is overestimated, but not so that of the "frontal" area.

(11) *The* "*Insular*" *Cortex.*

The "insular" cortex is all of a primitive type, that in front of the sulcus centralis has some olfactory characters, that behind temporal. It bears no resemblance to the "speech" cortex.

The paper was illustrated by lantern slides, drawings and casts.

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